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PASSMEDICINE

ENDOCRINOLOGY

Internal medicine

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2025

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June 29, 2025

← Question 1 of 342 →

A 20-year-old female presented to the accident and emergency department with severe abdominal pain, vomiting and lethargy. On further questioning she stated that she had been generally unwell for the last four months during which time she lost 10 Kg in weight and had been tired all the time.

Last month she has been diagnosed with hypothyroidism and was prescribed levothyroxine 50 mcg daily.

Her mother and sister have hypothyroidism and take thyroxine. On examination, she looks unwell and dehydrated.

Her pulse is 105 beats per minute and blood pressure is 70/40 mmHg

Her temperature is 37.6°C and BMI is 19 kg/m^2. Cardiovascular, respiratory and abdominal examination were normal. Investigations done last month showed:

Hb	9.5 g/dl
MCV	105 fl
Platelets	190 * 10^9/l
WBC	4.5 * 10^9/l

Serum free T4	8.5 pmol/l
Serum TSH	5.5 mU/l

While awaiting new investigations, what is the most appropriate immediate treatment for this patient?

○ Intravenous glucose 10% ×

○ Intravenous normal saline ×

○ Intravenous normal saline and antibiotics ✕

○ Intravenous normal saline and hydrocortisone ✕

○ Intravenous thyroxine ✕

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **0%**

1 -

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Dr. Jamil Alrajhi

← Question 1 of 342 ✓ →

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Hb	9.5 g/dl
MCV	105 fl
Platelets	$190 * 10^9$/l
WBC	$4.5 * 10^9$/l

Serum free T4	8.5 pmol/l
Serum TSH	5.5 mU/l

While awaiting new investigations, what is the most appropriate immediate treatment for this patient?

Intravenous glucose 10%	1%
Intravenous normal saline	6%

Intravenous normal saline and antibiotics	1%
[✓ Intravenous normal saline and hydrocortisone]	89%
Intravenous thyroxine	2%

This patient presented with Addisonian crisis (abdominal pain, vomiting, dehydration and hypotension). She has been complaining of tiredness and weight loss (which are features of Addisons disease) for four months but what precipitated the crisis is the thyroxine given for the presumed hypothyroidism.

Actually, a slightly raised TSH and a decreased T4 are features of primary hypoadrenalism and do not necessarily indicate frank hypothyroidism.

This is a medical emergency and should be treated immediately with intravenous normal saline and hydrocortisone. Thyroxine should not be given as it will exacerbate the condition.

Her low haemoglobin and high MCV may point towards pernicious anaemia which is an autoimmune disease seen sometimes in association with Addisons disease.

👍 👎 Discuss (8) Improve

Next question

Addisonian crisis

Causes

- sepsis or surgery causing an acute exacerbation of chronic insufficiency (e.g. Addison's, hypopituitarism)
- adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcaemia)
- steroid withdrawal (most common precipitant)

- inadequate 'sick day rule' management or intercurrent illness (commonly gastroenteritis) in patients with underlying Addison's disease

Signs and symptoms
- confusion, syncope, lethargy, convulsions
- hypotension, tachycardia, hypothermia
- hyponatraemia, hyperkalaemia, hypoglycaemia
- abdominal pain or cramps

Management
- hydrocortisone 100 mg IM or IV followed by 200 mg per 24 hours as a continuous infusion or 50 mg IV or IM every 6 hours (taper according to response as the patient becomes stable)
- 1 litre of normal saline infused over 30–60 minutes, then a further 4–6 litres over the first 24 hours if required; include dextrose if hypoglycaemic
- no fludrocortisone is required acutely as high-dose cortisol exerts a weak mineralocorticoid effect
- once stable, convert to oral replacement after approximately 24 hours, reducing to maintenance dosing over 3–4 days
- identify and treat the precipitating cause (e.g. infection)
- educate patients (and carers) about 'sick day rules' and encourage use of steroid alert cards or bracelets to prevent future crises

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 8 👎 2

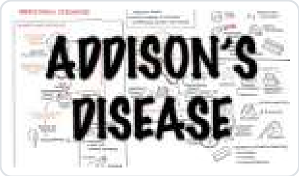
2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 6 👎 0



Addison's disease

Armando Hasudungan - YouTube 👍 4 👎 0

Report broken media

Score: **100%**

1 ✓

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A 52-year-old man presents to the eye clinic with a 3-month history of progressive visual disturbance. He describes difficulty seeing objects in the periphery of his vision, particularly when driving. He also reports persistent headaches and increased fatigue over the past 6 months. On examination, visual field testing reveals bitemporal hemianopia. An MRI brain with contrast is performed, showing a 2.5 cm pituitary mass with suprasellar extension compressing the optic chiasm. Blood tests are performed:

TSH	0.2 mU/L	(0.4-4.0)
Free T4	9.2 pmol/L	(12.0-22.0)
Prolactin	420 mU/L	(86-324)
Cortisol (9am)	120 nmol/L	(170-500)
LH	1.1 U/L	(1.5-9.3)
FSH	1.3 U/L	(2.0-12.0)
IGF-1	18 nmol/L	(14-30)

What is the most appropriate next step in management?

- ○ Cabergoline therapy ×
- ● Transsphenoidal surgery ×
- ○ Radiotherapy ×
- ○ Hormone replacement therapy only ×
- ○ Watchful waiting with repeat MRI in 3 months ×

Submit answer

Reference ranges

Score: **100%**

1	✓
2	-

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Dr. Jamil Alrajhi

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FSH	1.3 U/L	(2.0-12.0)
IGF-1	18 nmol/L	(14-30)

What is the most appropriate next step in management?

Cabergoline therapy	2%
[✓ Transsphenoidal surgery]	98%
Radiotherapy	0%
Hormone replacement therapy only	0%
Watchful waiting with repeat MRI in 3 months	0%

Non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - transsphenoidal surgery is therefore the first-line treatment

Important for me Less important

Transsphenoidal surgery is the most appropriate first-line treatment for this patient. The clinical presentation strongly suggests a non-functioning pituitary macroadenoma (2.5 cm in size with suprasellar extension) causing significant mass effect. The patient has clear evidence of compressive symptoms including bitemporal hemianopia due to optic chiasm compression, headaches, and laboratory evidence of hypopituitarism affecting multiple hormonal axes (low TSH/T4, low cortisol, low gonadotropins). The moderately elevated prolactin level is likely due to stalk effect (compression of the pituitary stalk) rather than a true prolactinoma, as the elevation is relatively modest for the size of the tumour. Transsphenoidal surgery would address both the visual symptoms by decompressing the optic chiasm and potentially improve pituitary function by removing the compressive mass. This approach is consistent with UK guidelines for management of non-functioning pituitary macroadenomas with visual compromise, where surgical decompression is the priority intervention.

Cabergoline therapy would be the first-line treatment for prolactinomas, but this patient's clinical picture is not consistent with a primary prolactinoma. The modest prolactin elevation (420 mU/L) is disproportionately low for a 2.5 cm tumour if it were a true prolactinoma (which would typically show prolactin levels >2000 mU/L for a tumour of this size). The pattern of multiple hormonal deficiencies and significant mass effect is more consistent with a non-functioning adenoma causing stalk compression, which would not respond adequately to dopamine agonist therapy.

Radiotherapy is not appropriate as first-line treatment for pituitary adenomas. It is typically reserved for residual or recurrent tumour after surgery, or in cases where surgery is contraindicated. Radiotherapy carries risks of further pituitary dysfunction and potential damage to surrounding structures, making it a second-line or adjunctive treatment rather than the initial approach.

Hormone replacement therapy only would address the hormonal deficiencies but would not treat the underlying tumour or relieve the compressive symptoms. This patient has significant visual field defects that require urgent decompression to prevent permanent visual loss. Hormone replacement would be an important adjunctive therapy but is insufficient as the sole treatment given the mass effect symptoms.

Watchful waiting with repeat MRI in 3 months is inappropriate given the presence of significant visual field defects and multiple hormonal deficiencies. The optic chiasm compression requires urgent intervention to prevent permanent visual loss. Delaying treatment in a patient with established visual compromise and panhypopituitarism would risk further neurological deterioration and is not consistent with standard of care for symptomatic pituitary macroadenomas.

👍 | 👎 | 💬 Discuss | Improve

Next question

Pituitary adenoma ★

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:
- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due

to excess prolactin)
- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
  - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
  - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).
  - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
  - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone)

    - and neuromodulators like pasireotide
- transsphenoidal surgery
    - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
    - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
    - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
    - indicated for residual or recurrent tumours post-surgery

+ Q 123

Next question

Textbooks

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Patient.info 👍 20 👎 11

Pituitary tumours review

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Score: **100%**

1 ✓

2 ✓

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A 49-year-old man presents with feeling under the weather. On further questioning he reports that he has been feeling tired and weak for the past few weeks and his wife has noticed that he as lost some weight, although he states that his appetite has not decreased and if anything he is feeling more thirsty and going to the toilet several times a night. Over the last couple of days he has noticed a rash develop around his groin, which is now present on his buttocks. On examination, there are patches of red with irregular borders and crusting.

A fasting blood test is arranged and reveals a blood glucose of 9.2 mm/l.

What is the next most appropriate investigation?

- ○ Plasma insulin level ×
- ○ Tissue transglutaminase antibody (TTA) test ×
- ○ Plasma glucagon level ×
- ○ Skin biopsy ×
- ○ Plasma zinc level ×

Submit answer

Reference ranges

Score: **100%**

1	✓
2	✓
3	-

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A fasting blood test is arranged and reveals a blood glucose of 9.2 mm/l.

What is the next most appropriate investigation?

Plasma insulin level	12%
Tissue transglutaminase antibody (TTA) test	22%
[✓ Plasma glucagon level]	52%
Skin biopsy	5%
Plasma zinc level	9%

This patient has a diagnosis of a glucagonoma. The polydipsia and polyuria, coupled with the fasting blood glucose result suggest diabetes and the rash is necrolytic migratory erythema a symptom which is the presenting problem in up to 70% of cases of glucagonoma.

Discuss (6) Improve

Next question

Glucagonoma ★

Glucagonomas are small tumours, almost always found in the pancreas, and frequently malignant. The tumours arise from the alpha cells of the pancreas.

They present with diabetes mellitus, venous thrombo-embolism and the classical rash of necrolytic migratory erythema - a red, blistering rash.

A serum level of glucagon >1000pg/ml usually suggests the diagnosis, imaging with CT scanning is also required.

Treatment options include surgical resection and octreotide.

+ Q 123

Next question

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Score: **100%**

1 ✓

2 ✓

3 ✓

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A 45-year-old woman develops agitation, high fever, sweating and atrial fibrillation with a heart rate of 160bpm following a CT pulmonary angiogram. She was recently seen in the emergency department and given a provisional diagnosis of pulmonary embolism and discharged with an urgent CT pulmonary angiogram and treatment dose low molecular weight heparin two days ago. She has a past medical history of breast cancer which has recently recurred with metastases to the liver, depression and COPD. She normally takes sertraline, letrozole, carbimazole and as needed salbutamol. Her original presentation to the emergency department was with shortness of breath which has now resolved. The CT scan has demonstrated no evidence of pulmonary embolism and clear lung fields. On examination, she is very agitated, has tremors, is very hot to touch and has ankle oedema. She has bilateral crepitations on chest auscultation. Her temperature is 40.1°C. Blood tests are pending. What is the most likely cause of her presentation?

- ○ Delayed autoimmune reaction secondary to contrast medium ×
- ○ Atypical pneumonia ×
- ○ Missed pulmonary embolism with right heart strain ×
- ○ Thyroid storm ×
- ○ Heparin induced thrombocytopenia ×

Submit answer

Reference ranges

Score: **100%**

1	✓
2	✓
3	✓
4	-

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Delayed autoimmune reaction secondary to contrast medium	8%
Atypical pneumonia	1%
Missed pulmonary embolism with right heart strain	1%
Thyroid storm	89%
Heparin induced thrombocytopenia	1%

Iodine in CT contrast media can precipitate thyrotoxicosis or thyroid storm

Important for me Less important

The correct answer is thyroid storm. The iodine in the CT contrast medium can trigger thyrotoxicosis and thyroid storm. The features of hyperpyrexia, agitation, confusion, AF with a fast ventricular rate in a patient with known hyperthyroidism following a triggering event, such as CT contrast, infection or surgery can thyrotoxicosis or thyroid storm. An autoimmune reaction is unlikely given the absence of rash and presence of fever and agitation. Pneumonia is unlikely without any radiological features on the CT scan and also with the absence of a cough, and missing pulmonary embolism is unlikely, but it is also unlikely to miss right heart strain on CT.

👍 👎 Discuss (6) Improve

Next question

Thyroid storm

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:
- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event

- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

B I A

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Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 8 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

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Score: **100%**

1 ✓
2 ✓
3 ✓
4 ✓

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← Question 5 of 342 ⚐ →

A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is noted to have non-visible haematuria during an annual review. He is currently feeling well and is asymptomatic. The urine dipstick showed blood ++, with no protein and no leucocytes. This result is repeated one week later.

His current medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin and pioglitazone.

Which one of the following drugs should be stopped whilst awaiting further investigations?

○ Aspirin ×
○ Ramipril ×
○ Atorvastatin ×
○ Metformin ×
○ Pioglitazone ×

Submit answer

Reference ranges ∨

Score: **100%**

1 ✓

2	✓
3	✓
4	✓
5	-

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Question 5 of 342 ✗

A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is noted to have non-visible haematuria during an annual review. He is currently feeling well and is asymptomatic. The urine dipstick showed blood ++, with no protein and no leucocytes. This result is repeated one week later.

His current medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin and pioglitazone.

Which one of the following drugs should be stopped whilst awaiting further investigations?

🟥	Aspirin	12%
	Ramipril	5%
	Atorvastatin	3%
	Metformin	5%
🟩	Pioglitazone	75%

Thiazolidinediones are associated with an increased risk of bladder cancer

Important for me Less important

The correct answer is **Pioglitazone**. Pioglitazone, an oral antidiabetic agent used in the management of type 2 diabetes mellitus, has been associated with an increased risk of bladder cancer. In the presence of non-visible haematuria (blood in urine), this medication should be discontinued until further investigations are completed.

Aspirin is not typically associated with haematuria and its use as antiplatelet therapy in a patient with ischemic heart disease should not be interrupted without a compelling reason.

Ramipril, an angiotensin-converting enzyme (ACE) inhibitor, is used for hypertension and cardiac conditions. While it can occasionally cause renal impairment leading to proteinuria, it does not typically cause haematuria.

Atorvastatin, a statin used for hyperlipidaemia and cardiovascular risk reduction, may have some side effects such as myopathy or liver dysfunction but it is not commonly associated with haematuria.

Finally, **Metformin**, another oral antidiabetic agent, can lead to lactic acidosis in cases of renal insufficiency but it does not usually cause haematuria. Therefore, there would be no need to stop metformin based on the given clinical scenario.

👍 | 👎 | Discuss (3) | Improve

Next question

Thiazolidinediones ★

Thiazolidinediones are a class of agents used in the treatment of type 2 diabetes mellitus. They are agonists to the PPAR-gamma receptor and reduce peripheral insulin resistance. Rosiglitazone was withdrawn in 2010 following concerns about the cardiovascular side-effect profile.

The PPAR-gamma receptor is an intracellular nuclear receptor. It's natural ligands are free fatty acids and it is thought to control adipocyte differentiation and function.

Adverse effects

- weight gain
- liver impairment: monitor LFTs
- fluid retention - therefore contraindicated in heart failure. The risk of fluid retention is increased if the patient also takes insulin
- recent studies have indicated an increased risk of fractures

- bladder cancer: recent studies have shown an increased risk of bladder cancer in patients taking pioglitazone (hazard ratio 2.64)

+ Q 123

Next question

Textbooks

High-yield textbook

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Links

NICE 👍 12 👎 7

2022 Type 2 diabetes guidelines

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Score: **80%**

1 ✓
2 ✓
3 ✓
4 ✓

5 ✗

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A 33-year-old woman presents to the endocrinology clinic for review. She has a past medical history of Graves' disease which was treated with radioiodine treatment. This finished one year ago. Following this, she has retained a clinically and biochemically euthyroid state. She originally presented with weight loss and insomnia, and these symptoms have not re-occurred. Her GP has requested blood tests prior to the appointment which has shown a TSH of 2.6 and a free T4 of 8.2mg/dl. She wanted to have a review as she has recently stopped taking oral contraceptive tablets and is planning to become pregnant. What is the most appropriate plan?

- ○ Advise to avoid pregnancy ×
- ○ Measure serum thyroid-stimulating hormone receptor antibodies ×
- ○ Arrange for US scan of neck ×
- ○ Repeat TSH and free T4 in one year's time ×
- ○ Measure thyrotrophin receptor stimulating antibodies ×

Submit answer

Reference ranges

Score: **80%**

1 ✓

2	✓
3	✓
4	✓
5	✗
6	-

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Advise to avoid pregnancy	6%
[✓ Measure serum thyroid-stimulating hormone receptor antibodies]	53%
Arrange for US scan of neck	4%
Repeat TSH and free T4 in one year's time	14%
Measure thyrotrophin receptor stimulating antibodies	23%

Pregnant woman with a history of Grave's disease should have thyroid stimulating hormone binding antibody titres measured even if euthyroid as the antibodies can cross the placental barrier

Important for me   Less important

This is a patient with a past medical history of Graves' disease who is clinically and biochemically euthyroid who is planning pregnancy. It is important, even with no biochemical evidence of hyperthyroidism, to exclude the serum presence of thyroid-stimulating hormone receptor antibodies as these can cross the placenta and cause foetal problems. If they were positive, then treatment should be initiated to control the antibody levels, despite the normal TSH and T4. Thyrotrophin receptor stimulating antibodies should be checked in hyperthyroidism at 30-36 weeks gestation to help assess the risk of neonatal thyroid problems. There is no reason she should not become pregnant.

👍 👎 Discuss (19) Improve

Next question

Pregnancy: thyroid problems

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems

- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points

- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
  - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

B I A T↕

Textbooks

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Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

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Score: **83.3%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓

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A 74-year-old woman with a history of diverticular disease and Graves' disease presents with lower abdominal pain, fever and bloody diarrhoea. She has a CT of the abdomen with contrast, which confirms diverticulitis. This responds well to treatment, and she is discharged.

The patient is readmitted three weeks later in fast atrial fibrillation. On assessment, she reports a one-week history of palpitations, non-bloody diarrhoea and feeling hot all the time. She continues to smoke 5 cigarettes per day and denies recent coryzal symptoms.

On examination, she has an irregular pulse of 134 bpm and a BP of 132/88 mmHg. Her abdomen is soft and non-tender, with active bowel sounds. A non-tender goitre is present.

Her investigations:

Thyroid stimulating hormone (TSH)	< 0.05 mU/L	(0.5-5.5)
Free thyroxine (T4)	24 pmol/L	(9.0 - 18)

What is the most likely cause of her presentation?

- ○ Administration of contrast
- ○ De Quervain’s thyroiditis
- ○ Further episode of diverticulitis
- ○ Riedel's thyroiditis
- ○ Smoking

Submit answer

Reference ranges

Score: **83.3%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	-

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A 74-year-old woman with a history of diverticular disease and Graves' disease presents with lower abdominal pain, fever and bloody diarrhoea. She has a CT of the abdomen with contrast, which confirms diverticulitis. This responds well to treatment, and she is discharged.

The patient is readmitted three weeks later in fast atrial fibrillation. On assessment, she reports a one-week history of palpitations, non-bloody diarrhoea and feeling hot all the time. She continues to smoke 5 cigarettes per day and denies recent coryzal symptoms.

On examination, she has an irregular pulse of 134 bpm and a BP of 132/88 mmHg. Her abdomen is soft and non-tender, with active bowel sounds. A non-tender goitre is present.

Her investigations:

Thyroid stimulating hormone (TSH)	< 0.05 mU/L	(0.5-5.5)
Free thyroxine (T4)	24 pmol/L	(9.0 - 18)

What is the most likely cause of her presentation?

[✓ Administration of contrast]	67%
De Quervain’s thyroiditis	14%
Further episode of diverticulitis	1%
Riedel's thyroiditis	11%
Smoking	7%

Thyrotoxicosis may develop in patients following the administration of contrast, particularly if a background of thyroid disease

Important for me Less important

Administration of contrast is the correct answer here. This can precipitate thyrotoxicosis in patients with a history of previous thyroid disease. IV contrast results in a load of iodine to the thyroid, resulting in increased secretion of thyroid hormones and hyperthyroidism, which can develop over 2-12 weeks.

De Quervain's thyroiditis often follows an upper respiratory viral illness and usually presents with a painful goitre. In this case, the patient denies coryzal symptoms and has a non-tender goitre, making this diagnosis less likely.

Though a **further episode of diverticulitis** is possible, the vignette suggests a clinical resolution, and this diagnosis would be unlikely with a soft, non-tender abdomen.

Patients with **Riedel's thyroiditis** classically present with clinical and biochemical features consistent with hypothyroidism, which is not the case here.

While the patient has a history of **smoking** , her intake has not drastically increased. Furthermore, smoking induces mild hyperthyroidism, so this is unlikely to be the cause of her presentation in this case.

Discuss Improve

Next question

Thyrotoxicosis: causes and investigation

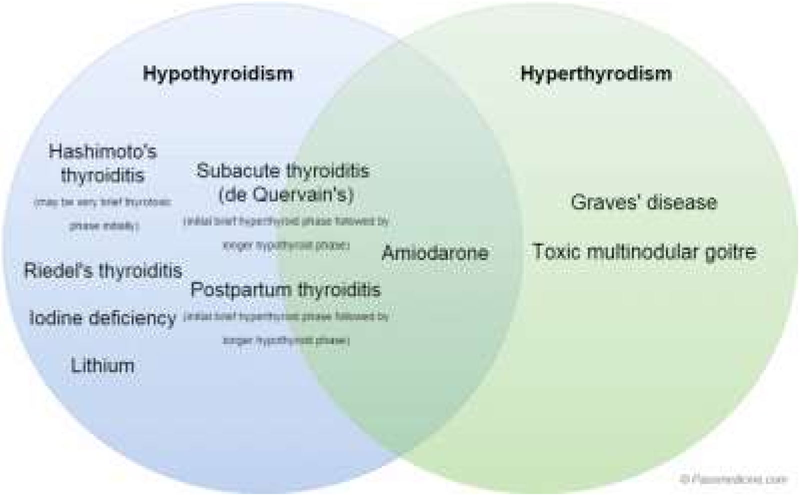
Graves' disease accounts for around 50-60% of cases of thyrotoxicosis.

Causes
- Graves' disease
- toxic nodular goitre
- acute phase of subacute (de Quervain's) thyroiditis
- acute phase of post-partum thyroiditis

- acute phase of Hashimoto's thyroiditis (later results in hypothyroidism)
- amiodarone therapy
- contrast
  - rare, more commonly occurs in elderly patients with pre-existing thyroid disease (e.g. multinodular goitre, Graves')
  - patients with existing thyrotoxicosis should not receive iodinated contrast medium
  - administration of iodinated contrast results in a large iodine load to the thyroid → hyperthyroidism developing over 2-12 weeks

Investigation

- TSH down, T4 and T3 up
- thyroid autoantibodies
- other investigations are not routinely done but includes isotope scanning


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
Amiodarone
Graves' disease
Toxic multinodular goitre
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 2 👎 4

Hyperthyroidism guidelines

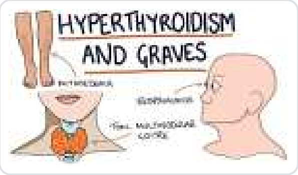
Royal College of Physicians 👍 7 👎 3

2017 Thyrotoxicosis â€“ investigation and management

Suggest link

Report broken link

Media


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 2 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube 👍 0 👎 0

Report broken media

Score: **85.7%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓

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← Question 8 of 342 →

A 17-year-old man with a known history of type 1 diabetes mellitus presents to the Emergency Department with nausea, vomiting, and abdominal pain. On examination, he is alert, oriented, and clinically dehydrated. His vital signs reveal a blood pressure of 90/60 mmHg, a pulse rate of 115 bpm, and an oxygen saturation of 97% on room air.

His laboratory investigations are as follows:

Hb	150 g/L	(135–180)
Platelets	170×10^9/L	(150–400)
WBC	12×10^9/L	(4.0–11.0)
Na^+	132 mmol/L	(135–145)
K^+	4.5 mmol/L	(3.5–5.0)
Bicarbonate	9 mmol/L	(22–29)
Urea	3 mmol/L	(2.0–7.0)
Creatinine	90 µmol/L	(55–120)
pH	7.15	(7.35–7.45)
Random blood glucose	23 mmol/L	(3.9–7.8 mmol/L)

1 litre of intravenous saline is given over 1 hour, and insulin infusion is initiated.

how you manage her potassium according to the JBDS guidelines?

○ 10 mmol of potassium per litre of saline over the next 2 hours ×

● 20 mmol of potassium per litre of saline over the next 2 hours ×

○ 40 mmol of potassium per litre of saline over the next 2 hours ×

○ Administer potassium replacement only if $K^+ < 3.5$ mmol/L ×

○ Hold potassium replacement until urine output is established ×

Submit answer

Reference ranges

Score: **85.7%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	-

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1 litre of intravenous saline is given over 1 hour, and insulin infusion is initiated.

how you manage her potassium according to the JBDS guidelines?

	10 mmol of potassium per litre of saline over the next 2 hours	7%
🟥	20 mmol of potassium per litre of saline over the next 2 hours	35%
🟩	40 mmol of potassium per litre of saline over the next 2 hours	37%
	Administer potassium replacement only if $K^+ < 3.5$ mmol/L	17%

Hold potassium replacement until urine output is established 4%

Diabetic ketoacidosis: 40 mmol of potassium should be added to a litre of saline if the potassium is within 3.5-5.5mmol/L

Important for me Less important

The correct answer is: **40 mmol of potassium per litre of saline over the next 2 hours**. In diabetic ketoacidosis (DKA), when serum potassium is between 3.5 and 5.5 mmol/L, current guidelines recommend adding 40 mmol/L of potassium to each litre of intravenous saline. This practice helps prevent insulin-induced hypokalaemia by maintaining serum potassium within the target range.

10 mmol of potassium per litre of saline over the next 2 hours is incorrect. Administering only 10 mmol/L of potassium is inadequate for preventing hypokalaemia once insulin therapy has commenced. Insulin promotes a rapid intracellular shift of potassium, and such a low dose would not sufficiently maintain appropriate serum levels.

20 mmol of potassium per litre of saline over the next 2 hours, while more effective than 10 mmol/L, remains below the recommended dose according to national guidelines for DKA management. Evidence indicates that this amount does not reliably compensate for the significant intracellular shift caused by insulin administration and fluid resuscitation, thereby increasing the risk of hypokalaemia.

Administer potassium replacement only if $K^+ < 3.5$ mmol/L is incorrect. This approach is overly conservative and may be unsafe in clinical practice. Insulin therapy results in a prompt intracellular movement of potassium, which can precipitate severe hypokalaemia if replacement is delayed. Guidelines advise commencing potassium supplementation if K^+ is less than 5.5 mmol/L, and withholding insulin only if K^+ falls below approximately 3.0–3.3 mmol/L.

Withhold potassium replacement until urine output is established is also incorrect. While monitoring urine output remains important, most patients presenting with DKA retain adequate renal function upon arrival at the Emergency Department. Delaying potassium replacement until urine output has been confirmed increases the risk of worsening hypokalaemia due to ongoing insulin-driven shifts into cells. Best practice involves early initiation of potassium replacement alongside close monitoring of both serum levels and urine output.

Clinical Pearl: In diabetic ketoacidosis, timely administration of intravenous fluids containing 40 mmol/L potassium chloride constitutes standard care for adults with serum potassium between 3.5 and 5.5 mmol/L (JBDS-02 DKA Guideline, March 2023). Early correction reduces both the incidence and severity of hypokalaemia as well as associated cardiac complications.

Reference: Joint British Diabetes Societies for Inpatient Care (JBDS-IP) – The Management of Diabetic Ketoacidosis in Adults (JBDS-02), March 2023.

JBDS DKA Guideline

👍 👎 Discuss Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed

insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management
- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low

    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to

monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

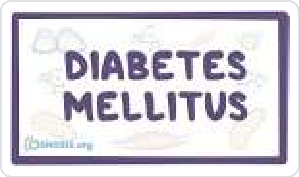
Report broken link

Media

Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



[Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms]

Osmosis - YouTube

👍 9 👎 3

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Score: **75%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗

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Question 9 of 342

A 55-year-old woman presents with several months of progressive facial changes and an increase in shoe and ring size. She has also developed new-onset loud snoring. Her past medical history includes well-controlled hypertension for two years. Her husband reports that her appearance has changed, particularly around the jaw and nose. On examination, her facial features are coarse, and she has mild prognathism. Blood pressure is 145/90 mmHg.

What is the most appropriate initial investigation?

- ○ Chest and abdomen CT ×
- ○ MRI of the pituitary gland ×
- ○ Morning growth hormone (GH) measurements ×
- ○ Oral glucose tolerance test with GH suppression ×
- ○ Serum IGF-1 level ×

Submit answer

Reference ranges

Score: **75%**

1 ✓
2 ✓
3 ✓

4	✓
5	✗
6	✓
7	✓
8	✗
9	-

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Question 9 of 342 ✓

A 55-year-old woman presents with several months of progressive facial changes and an increase in shoe and ring size. She has also developed new-onset loud snoring. Her past medical history includes well-controlled hypertension for two years. Her husband reports that her appearance has changed, particularly around the jaw and nose. On examination, her facial features are coarse, and she has mild prognathism. Blood pressure is 145/90 mmHg.

What is the most appropriate initial investigation?

Chest and abdomen CT	0%
MRI of the pituitary gland	3%
Morning growth hormone (GH) measurements	0%
Oral glucose tolerance test with GH suppression	16%
Serum IGF-1 level	81%

Serum IGF-1 levels are now the first-line test for acromegaly

Important for me Less important

The clinical presentation is highly suggestive of acromegaly. The patient has a two-year history of hypertension and reports progressive changes in facial appearance, increasing shoe size, and new-onset snoring, all hallmark features of excess GH secretion. Examination findings of coarse facial features and mild prognathism further support this diagnosis.

Serum insulin-like growth factor 1 (IGF-1) level is the correct first-line investigation for suspected acromegaly. Because growth hormone (GH) is secreted in a pulsatile fashion, random GH measurements are unreliable for screening. In contrast, IGF-1 levels remain relatively stable throughout the day and reflect integrated GH secretion, making them the preferred initial screening test according to current guidelines. IGF-1 reflects chronic GH excess, which causes bony/facial changes (e.g., prognathism, snoring) and hypertension in acromegaly.

MRI pituitary should be performed only after biochemical confirmation of acromegaly with elevated IGF-1 levels. Imaging is used to localise the source of GH excess, typically a pituitary adenoma.

Morning GH measurements are not recommended due to the pulsatile nature of GH secretion, which results in considerable intra-day variability and limits the utility of a single reading.

Chest and abdomen CT is indicated only if biochemical confirmation of acromegaly is present and pituitary imaging fails to identify a tumour. In such cases, ectopic GH or GHRH secretion (e.g., from neuroendocrine tumours) must be considered.

Oral glucose tolerance test with GH suppression is incorrect. It is used after IGF-1 is equivocal/elevated for biochemical confirmation. Lack of suppression of GH to < 1 µg/L following documented hyperglycemia during an oral glucose load of 75 g confirms the diagnosis of acromegaly.

👍 👎 Discuss Improve

Next question

Acromegaly: investigations ★

Growth hormone (GH) levels vary during the day and are therefore not diagnostic.

Serum IGF-1 levels have now overtaken the oral glucose tolerance test (OGTT) with serial GH measurements as the first-line test. The OGTT test is recommended to confirm the diagnosis if IGF-1 levels are raised.

The Endocrine Society guidelines suggest the following:

> *1.1 We recommend measurement of IGF-1 levels in patients with typical clinical manifestations of acromegaly, especially those with acral and facial features.*
> ...
> *1.5 In patients with elevated or equivocal serum IGF-1 levels, we recommend confirmation of the diagnosis by finding lack of suppression of GH to < 1 μg/L following documented hyperglycemia during an oral glucose load.*

Serum IGF-1 may also be used to monitor disease

Oral glucose tolerance test
- in normal patients GH is suppressed to < 2 mu/L with hyperglycaemia
- in acromegaly there is no suppression of GH
- may also demonstrate impaired glucose tolerance which is associated with acromegaly

A pituitary MRI may demonstrate a pituitary tumour.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 14 👎 3

2014 Acromegaly guidelines

Suggest link

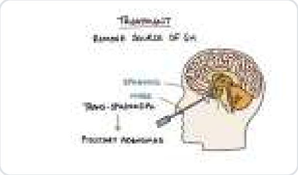
Report broken link

Media

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 1 👎 0



Understanding Acromegaly

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **77.8%**

1 ✓
2 ✓

3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓

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A 42-year-old woman presents with a three-week history of neck pain and swelling that worsens upon swallowing. She also reports fatigue, tremors, nervousness, occasional palpitations, and unintentional weight loss. Examination reveals blood pressure of 125/80 mmHg, heart rate of 104 beats per minute, and temperature of 37.8 °C. The thyroid gland is tender on palpation and enlarged without any palpable nodules. Laboratory results are as follows:

TSH	0.1 mIU/L ng/mL	(0.4 - 4.0)
Free T4	8 ng/dL	(0.7 - 1.9)
ESR	101 mm/hr	

Anti-thyroglobulin and anti-thyroperoxidase antibodies are mildly elevated, while radioactive iodine uptake is low.

What is the most likely diagnosis?

- ○ Acute infectious thyroiditis ×
- ○ Graves disease ×
- ○ Hashimoto thyroiditis ×
- ○ Solitary toxic nodule ×
- ○ Subacute granulomatous thyroiditis ×

Submit answer

Reference ranges

Score: **77.8%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	-

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Free T4	8 ng/dL	(0.7 - 1.9)
ESR	101 mm/hr	

Anti-thyroglobulin and anti-thyroperoxidase antibodies are mildly elevated, while radioactive iodine uptake is low.

What is the most likely diagnosis?

🟥	Acute infectious thyroiditis	34%
	Graves disease	6%
	Hashimoto thyroiditis	12%
	Solitary toxic nodule	1%
🟩	Subacute granulomatous thyroiditis	46%

Thyrotoxicosis with tender goitre = subacute (De Quervain's) thyroiditis

Important for me Less important

Subacute granulomatous thyroiditis, also known as de Quervain's thyroiditis, is the correct diagnosis. The patient presents with clinical and biochemical signs of thyrotoxicosis, as indicated by tachycardia, tremors, nervousness, low-grade fever, fatigue, weight loss, palpitations, suppressed TSH, and elevated free T4 levels. These symptoms, in conjunction with an enlarged tender thyroid gland and high ESR level, are indicative of de Quervain's thyroiditis. Typically following a viral infection (not specified in this scenario), this condition is characterised by an initial phase of thyrotoxicosis followed by hypothyroidism. Management often involves symptomatic relief with non-steroidal anti-inflammatory drugs (NSAIDs) and beta-blockers.

Acute infectious thyroiditis, or suppurative thyroiditis, is a rare and potentially serious condition usually seen in the elderly and immunocompromised individuals. Gram-positive bacterial infections mainly cause it. Symptoms include sudden-onset fever, chills, malaise, a tender, painful thyroid gland, and signs of hyperthyroidism. In severe cases, the infection causes compression on adjacent structures, leading to dysphagia and hoarseness of voice. It is important to note that de Quervain's thyroiditis has a gradual onset of symptoms and does not cause compression symptoms, making acute infectious thyroiditis less likely.

Graves' disease is not the appropriate diagnosis here. Despite the biochemical and clinical features of thyrotoxicosis, the absence of Graves' ophthalmopathy and dermopathy, specific anti-thyroid stimulating hormone receptor antibodies, and a low radioactive iodine uptake are inconsistent with this condition.

Hashimoto's thyroiditis is a relevant differential diagnosis; however, it typically presents with a painless goitre. In de Quervain's thyroiditis, mild elevations in anti-thyroglobulin and anti-thyroperoxidase antibodies may be observed in fewer than 25% of cases. By contrast, these antibody levels are significantly higher in Hashimoto's thyroiditis.

Solitary toxic nodule is not consistent with this case presentation. Such nodules are generally single, palpable and non-tender associated with thyrotoxicosis. In contrast to this condition, the thyroid gland is diffusely enlarged and tender with no palpable; the findings align with de Quervain's thyroiditis rather than a solitary toxic nodule.

👍 👎 Discuss (1) Improve

Next question

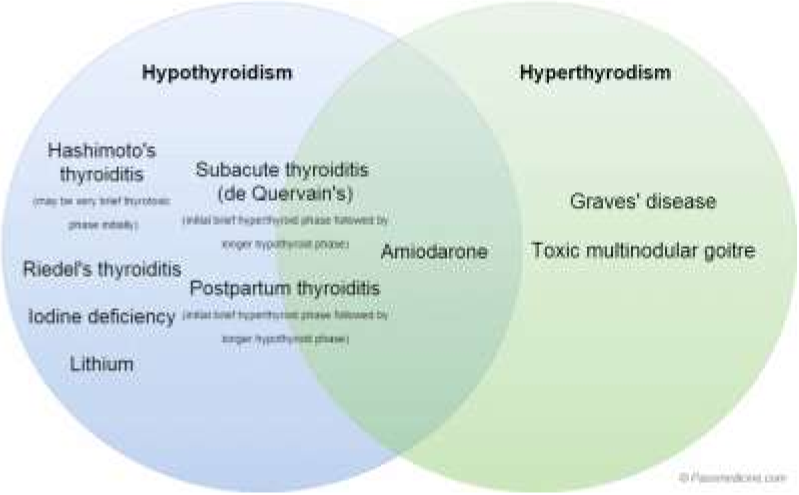
Subacute (De Quervain's) thyroiditis

Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations
- thyroid scintigraphy: globally reduced uptake of iodine-131

Management
- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
Iodine deficiency
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Lithium
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **70%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗

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Question 11 of 342 ✓

A 45-year-old patient presents to the endocrinology clinic. She presented to her GP with a small palpable thyroid nodule and the GP referred her to the endocrinology clinic because of this. She has a roughly 6mm thyroid nodule which is not painful or tender which she noticed incidentally. She has had no problems with her throat or systemic features. Following the GP appointment, she has had an ultrasound which demonstrates no evidence of malignant features. She has a past medical history of post-partum depression and fibroids. She takes no regular medications. What is the most appropriate plan?

Thyroid function tests	58%
US guided FNAC	36%
CT neck	0%
MRI neck	1%
Thyroid autoantibodies	4%

A new thyroid nodule does not need FNAC if the ultrasound features are benign

Important for me Less important

This patient has a small thyroid nodule. If the US features are benign in nature and there are no worrying features then a biopsy is not needed. A CT or MRI of the neck can be useful in the case of a large goitre of which the extent cannot be seen on ultrasound. Thyroid function tests would be useful to determine any level of thyroid dysfunction to exclude a toxic thyroid nodule. Autoantibodies can be useful in determining Grave's disease.

👍 👎 Discuss (10) Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	- Usually contain a mixture of papillary and colloidal filled follicles - Histologically tumour has papillary projections and pale empty nuclei - Seldom encapsulated - Lymph node metastasis predominate - Haematogenous metastasis rare
Follicular adenoma	- Usually present as a solitary thyroid nodule - Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	- May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. - Vascular invasion predominates - Multifocal disease raree
Medullary carcinoma	- C cells derived from neural crest and not thyroid tissue - Serum calcitonin levels often raised - Familial genetic disease accounts for up to 20% cases - Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	- Most common in elderly females - Local invasion is a common feature - Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

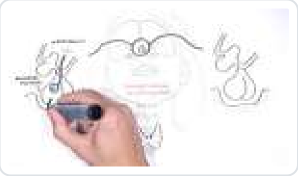
Suggest link Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0



Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **72.7%**

1 ✓
2 ✓
3 ✓
4 ✓
5 ✗
6 ✓
7 ✓
8 ✗
9 ✓
10 ✗
11 ✓

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Question 11 of 342 ✓

A 45-year-old patient presents to the endocrinology clinic. She presented to her GP with a small palpable thyroid nodule and the GP referred her to the endocrinology clinic because of this. She has a roughly 6mm thyroid nodule which is not painful or tender which she noticed incidentally. She has had no problems with her throat or systemic features. Following the GP appointment, she has had an ultrasound which demonstrates no evidence of malignant features. She has a past medical history of post-partum depression and fibroids. She takes no regular medications. What is the most appropriate plan?

Thyroid function tests	58%
US guided FNAC	36%
CT neck	0%
MRI neck	1%
Thyroid autoantibodies	4%

A new thyroid nodule does not need FNAC if the ultrasound features are benign

Important for me Less important

This patient has a small thyroid nodule. If the US features are benign in nature and there are no worrying features then a biopsy is not needed. A CT or MRI of the neck can be useful in the case of a large goitre of which the extent cannot be seen on ultrasound. Thyroid function tests would be useful to determine any level of thyroid dysfunction to exclude a toxic thyroid nodule. Autoantibodies can be useful in determining Grave's disease.

👍 👎 Discuss (10) Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

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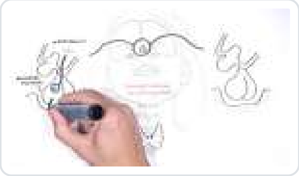
Suggest link Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0



Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **72.7%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓

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A 55-year-old man presents to the endocrine clinic. He was diagnosed five years ago with type 2 diabetes and is struggling to control his sugars. He is currently taking:

Metformin 1g BD
Glicazide 160mg BD
Sitagliptin 100mg OD

He is a bus driver and struggles to control his weight with his hectic shifts. Current BMI is 34 kg/m².

Investigations:

Serum creatinine	120 µmol/L (60-110)
Haemoglobin A1c	66 mmol/mol (8.2%)

What would be the most appropriate next step?

- ○ Canagliflozin ×
- ○ Glibenclamide ×
- ○ Increase metformin ×
- ○ Stop sitagliptin and add insulin ×
- ○ Stop sitagliptin and add exenatide ×

Submit answer

Reference ranges

Score: **72.7%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	-

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Back to top

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Metformin 1g BD
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Sitagliptin 100mg OD

He is a bus driver and struggles to control his weight with his hectic shifts. Current BMI is 34 kg/m².

Investigations:

Serum creatinine	120 µmol/L (60-110)
Haemoglobin A1c	66 mmol/mol (8.2%)

What would be the most appropriate next step?

Canagliflozin	14%
Glibenclamide	1%
Increase metformin	2%
Stop sitagliptin and add insulin	8%
[✓ Stop sitagliptin and add exenatide]	74%

Given the NICE guidance the most appropriate step would be to start this patient on exenatide. This patient is already of metformin, glicazide and sitagliptin and the blood sugar levels are not under control. His BMI is under 35 but insulin would make 'it much more difficult for you to do your job'.

👍 👎 Discuss (10) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

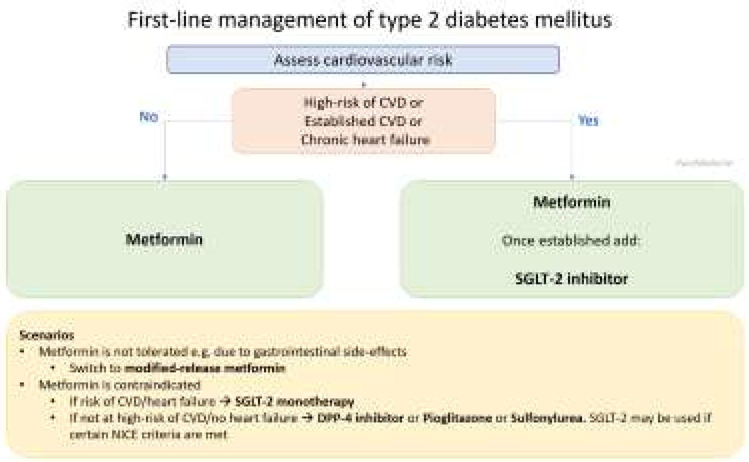
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

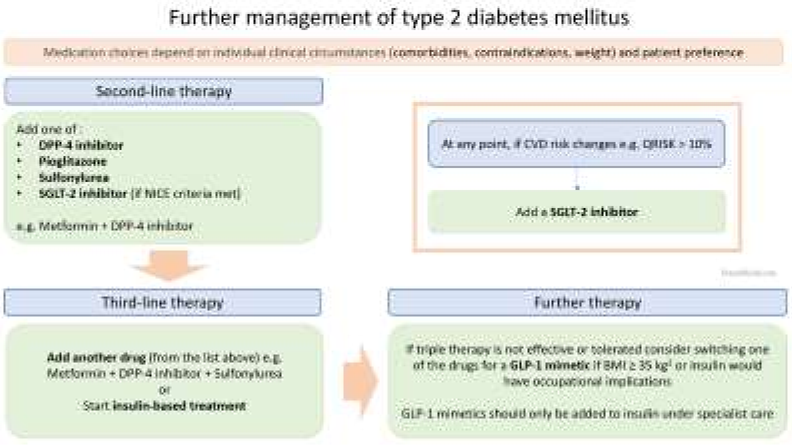
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:

    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

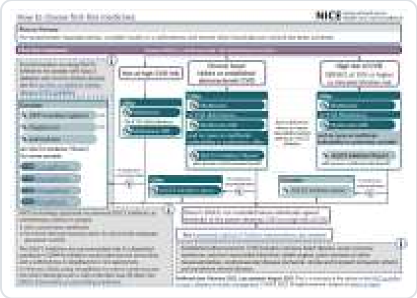
Starting insulin

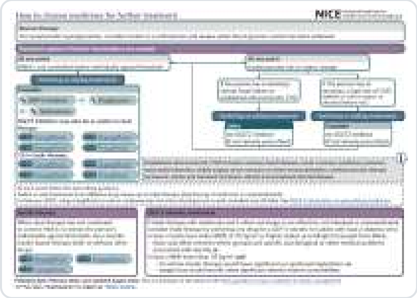
- metformin should be continued. In terms of other drugs NICE advice: '*Review the continued need for other blood glucose-lowering therapies*'
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

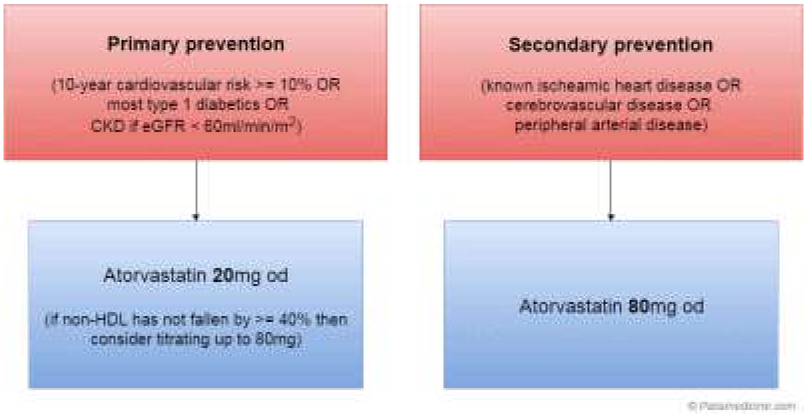
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

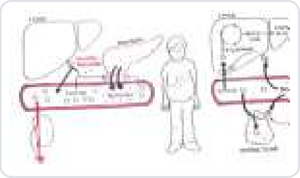
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

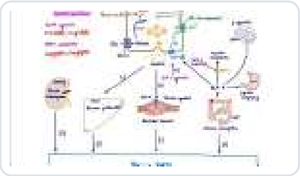
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **75%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗

9	✓
10	✗
11	✓
12	✓

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A 53-year-old female presents with 48 hours of general malaise. 30 years ago, she underwent a resection of a pituitary mass and has since been compliant on desmopressin, levothyroxine and hydrocortisone, up until her last dose earlier in the morning. She has no other past medical history. Her husband reports the patient to have had reduced oral intake for the past 2 days while she has been unwell. She has no reported head injuries, rigors or pyrexia. On examination, her GCS is E3 V2 M5. She is cool peripherally and a temperature demonstrates 33.4 degrees under her tongue. Her spot blood glucose is 2.2 mmol/l. Her blood pressure is 86/50 mmhg heart rate 110/min and sinus rhythm. Blood tests demonstrate a sodium of 158 mmol/l and potassium of 4.2 mmol/l. What is your first action(s)?

- ○ Send thyroid function test ×
- ○ Administer IV liothyronine ×
- ○ Send random cortisol ×
- ○ Administer IV hydrocortisone ×
- ○ CT head, blood culture, urine dip, HbA1c ×

Submit answer

Reference ranges

Score: **75%**

1	✓
2	✓
3	✓
4	✓
5	✕
6	✓
7	✓
8	✕
9	✓
10	✕
11	✓
12	✓
13	-

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Question 13 of 342 ✓

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Send thyroid function test	1%
Administer IV liothyronine	7%
Send random cortisol	1%
✓ Administer IV hydrocortisone	90%
CT head, blood culture, urine dip, HbA1c	1%

In the context of a sudden decline in any patient with long-term steroid use with hypotension, hypothermia and hypoglycaemia, you must treat presumptively for an Addisonian crisis. It must be remembered while the classic biochemical presentation of hyponatraemia and hyperkalaemia is frequently quoted in textbooks, this is frequently not the case in real life. While she may indeed be thyroid deficient, neither thyroid function tests nor intravenous free T3 is the most immediate treatment. A random cortisol, in the context of recent hydrocortisone in the community, is uninterpretable.

👍 👎 Discuss (6) Improve

Next question

Addison's disease: management

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:
- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:
- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards
- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 16

Addison's disease

Royal College of Physicians 👍 16 👎 8

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 1 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **76.9%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✓

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A 19-year-old woman presents to the emergency department drowsy and vomiting. She is accompanied by a friend who tells you she has been out drinking all day and has been vomiting for the last few hours.

Her speech is slurred and confused, she opens her eyes in response to her name and pushes you away in response to a painful stimulus. Heart rate is 100 beats per minute and regular, blood pressure is 100/60 mmHg, capillary glucose is 18 mmol/L, and a urine dip shows pH: 4, blood: trace, ketones: +++, protein: trace, nitrites: negative and leukocytes: negative.

Chest x-ray: Normal

Venous blood gas:

pH	7.27 (7.35-7.45)
Bicarbonate	10mmol/L (22-26)
Base excess	-10 (-2 to +2)
Sodium	135 mmol/L (137-144)
Potassium	2.9 mmol/L (3.5-4.9)
Chloride	99 mmol/L (95-107)

Serum Glucose: 21 mmol/L

What is the most important initial intervention?

○ Fixed rate intravenous insulin infusion (FRIII) ×

○ Intravenous calcium gluconate ×

○ Intravenous fluids ×

○ Sliding scale insulin ✕

○ Urgent CT head ✕

Submit answer

Reference ranges ⌄

Score: **76.9%**

1	✓
2	✓
3	✓
4	✓
5	✕
6	✓
7	✓
8	✕
9	✓
10	✕
11	✓
12	✓
13	✓
14	-

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Question 14 of 342 ✓

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Chest x-ray: Normal

Venous blood gas:

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Sodium	135 mmol/L (137-144)
Potassium	2.9 mmol/L (3.5-4.9)
Chloride	99 mmol/L (95-107)

Serum Glucose: 21 mmol/L

What is the most important initial intervention?

Fixed rate intravenous insulin infusion (FRIII)	12%
Intravenous calcium gluconate	1%
Intravenous fluids	86%
Sliding scale insulin	0%

Urgent CT head 0%

This woman fits the diagnostic criteria for diabetic ketoacidosis (DKA):

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO3-) < 15.0mmol/L and/or venous pH < 7.3

Diabetes UK guidance states that the most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by insulin administration.

The key benefits of fluid resuscitation in this context include:

- Recovery of circulatory volume
- Clearance of ketones and therefore improvement of acidosis
- Correction of electrolyte imbalance

Weight based fixed rate intravenous insulin infusion (FRIII) is now the recommended mode of insulin administration in DKA, over sliding scale.

DKA is a complication of type i diabetes (but can rarely complicate type ii diabetes). It can be the first presentation of type i diabetes, result from poor diabetic control or be precipitated by another factor such as infection.

Source:
diabetes UK, guidelines
https://www.diabetes.org.uk/Documents/About%20Us/What%20we%20say/Management
of-DKA-241013.pdf

👍 👎 Discuss (6) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours

Fluid	Volume
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

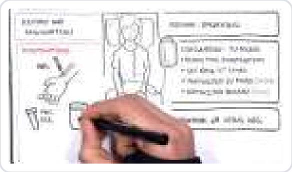
Links

Joint British Inpatient Diabetes Societies Inpatient Care Group 👍 33 👎 23

The Management of Diabetic ketoacidosis in adults

Suggest link Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2

DIABETES
MELLITUS
Osmosis.org

Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **78.6%**

1 ✓
2 ✓
3 ✓
4 ✓
5 ✗
6 ✓
7 ✓
8 ✗

9	✓
10	✗
11	✓
12	✓
13	✓
14	✓

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A 45-year-old gentleman is admitted to the psychiatry ward with a two day history of visual hallucinations. His past medical history is relevant for renal transplant last year, indicated because of membranous glomerulonephritis. He is on an established regime of immunosuppressants.

On examination, there is no evidence of focal neurology. Cranial nerves I to XII are normal, with equal and reactive pupils. There are no cerebellar signs. Plantars are equivocal on the left and downgoing on the right. Abbreviated mental test score (AMTS) is 8/10. An urgent CT head is unremarkable.

Which medication may be implicated as a cause of this patient's presentation?

- ○ Mycophenolate mofetil ×
- ○ Azathioprine ×
- ○ Prednisolone ×
- ○ Tacrolimus ×
- ○ Ciclosporin ×

Submit answer

Reference ranges

Score: **78.6%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✓
14	✓
15	-

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Question 15 of 342 ✓

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Which medication may be implicated as a cause of this patient's presentation?

Mycophenolate mofetil	12%
Azathioprine	2%
Prednisolone	48%
Tacrolimus	25%
Ciclosporin	13%

This patient is likely to be experiencing steroid-induced psychosis, and therefore, prednisolone is the correct answer. Most immunosuppressants have side-effects, and it is important to be aware of common associations. In addition to psychosis, steroids such as prednisolone may cause weight gain, secondary diabetes, osteoporosis and myopathy.

Ciclosporin and tacrolimus are both calcineurin inhibitors. Ciclosporin is associated with nephrotoxicity, gum hypertrophy and hirsutism.

Tacrolimus may cause tremor and secondary diabetes.

Mycophenolate mofetil and azathioprine are both anti-metabolites. Mycophenolate mofetil is associated with mucositis.

Azathioprine is steroid-sparing but can induce rashes, hepatitis and pancreatitis.

👍 👎 Discuss (10) Improve

Next question

Corticosteroids ★

Corticosteroids are amongst the most commonly prescribed therapies in clinical practice. They are used both systemically (oral or intravenous) or locally (skin creams, inhalers, eye drops, intra-articular). They augment and in some cases replace the natural glucocorticoid and mineralocorticoid activity of endogenous steroids.

The relative glucocorticoid and mineralocorticoid activity of commonly used steroids is shown below:

Minimal glucocorticoid activity, very high mineralocorticoid activity,	**Glucocorticoid activity, high mineralocorticoid activity,**	**Predominant glucocorticoid activity, low mineralocorticoid activity**	**Very high glucocorti activity, m mineraloc activity**
Fludrocortisone	Hydrocortisone	Prednisolone	Dexameth Betmethas

Side-effects

The side effects of corticosteroids are numerous and represent the single greatest limitation on their usage. Side-effects are more common with systemic and prolonged therapy.

Glucocorticoid side-effects
- endocrine
    - impaired glucose regulation
    - increased appetite/weight gain
    - hirsutism
    - hyperlipidaemia
- Cushing's syndrome
    - moon face
    - buffalo hump
    - striae
- musculoskeletal
    - osteoporosis
    - proximal myopathy
    - avascular necrosis of the femoral head
- immunosuppression
    - increased susceptibility to severe infection
    - reactivation of tuberculosis
- psychiatric
    - insomnia
    - mania
    - depression
    - psychosis
- gastrointestinal
    - peptic ulceration
    - acute pancreatitis
- ophthalmic
    - glaucoma
    - cataracts
- suppression of growth in children
- intracranial hypertension
- neutrophilia

Mineralocorticoid side-effects
- fluid retention
- hypertension

Selected points on the use of corticosteroids:

- patients on long-term steroids should have their doses doubled during intercurrent illness
- longer-term systemic corticosteroids suppress the natural production of endogenous steroids. They should therefore not be withdrawn abruptly, as this may precipitate an Addisonian crisis
- the BNF suggests gradual withdrawal of systemic corticosteroids if patients have:
    - received more than 40mg prednisolone daily for more than one week
    - received more than 3 weeks of treatment
    - recently received repeated courses

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **80%**

1 ✓
2 ✓
3 ✓

4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✓
14	✓
15	✓

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A 48-year-old man who was diagnosed with type 2 diabetes mellitus presents for review. During his annual review he was noted to have the following results:

Total cholesterol	5.3 mmol/l
HDL cholesterol	1.0 mmol/l
LDL cholesterol	3.1 mmol/l
Triglyceride	1.7 mmol/l
HbA1c	6.4%

A QRISK2 score is calculated showing that he has a 12% 10-year risk of developing cardiovascular disease. His current medication is metformin 500mg tds. According to recent NICE guidelines, what is the most appropriate action?

- ○ Simvastatin 40mg on ×
- ○ Lifestyle advice, repeat lipid profile in 3 months ×
- ○ Atorvastatin 40mg on ×
- ● Atorvastatin 20mg on ×
- ○ Increase his metformin slowly to 1g tds ×

Submit answer

Reference ranges

Score: **80%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
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12	✓
13	✓
14	✓
15	✓
16	-

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HbA1c	6.4%

A QRISK2 score is calculated showing that he has a 12% 10-year risk of developing cardiovascular disease. His current medication is metformin 500mg tds. According to recent NICE guidelines, what is the most appropriate action?

Simvastatin 40mg on	2%
Lifestyle advice, repeat lipid profile in 3 months	6%
Atorvastatin 40mg on	18%
[✓ Atorvastatin 20mg on]	73%
Increase his metformin slowly to 1g tds	1%

NICE recommend the following when considering the use of statins in patients with type 2 diabetes mellitus:

> *Offer atorvastatin 20 mg for the primary prevention of CVD to*

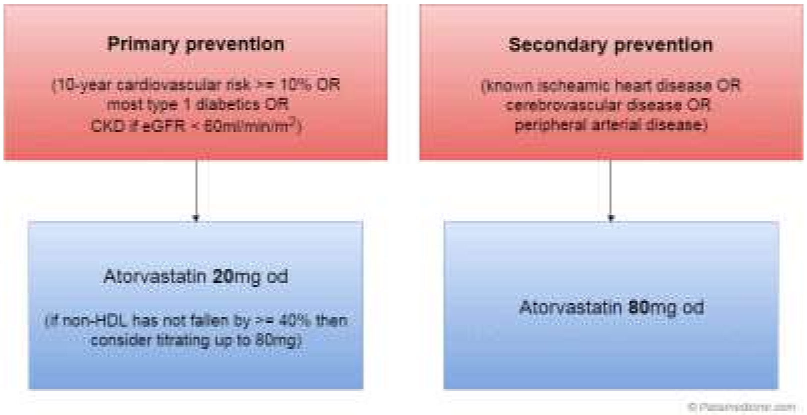
> *people with type 2 diabetes who have a 10% or greater 10-year risk of developing CVD.*

Discuss (3) | Improve

Next question

Hyperlipidaemia: management

In 2014 NICE updated their guidelines on lipid modification. This proved highly controversial as it meant that we should be recommending statins to a significant proportion of the population over the age of 60 years. Anyway, the key points of the new guidelines are summarised below.


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Primary prevention - who and how to assess risk

A systematic strategy should be used to identify people aged over 40 years who are likely to be at high risk of cardiovascular disease (CVD), defined as a 10-year risk of **10%** or greater.

NICE recommend we use the **QRISK2** CVD risk assessment tool for patients aged <= 84 years. Patients >= 85 years are at high risk of CVD due to their age. QRISK2 should not be used in the following situations as there are more specific guidelines for these patient groups:

- type 1 diabetics
- patients with an estimated glomerular filtration rate (eGFR) less than 60 ml/min and/or albuminuria
- patients with a history of familial hyperlipidaemia

NICE suggest QRISK2 may underestimate CVD risk in the following population groups:

- people treated for HIV
- people with serious mental health problems
- people taking medicines that can cause dyslipidaemia such as antipsychotics, corticosteroids or immunosuppressant drugs
- people with autoimmune disorders/systemic inflammatory disorders such as systemic lupus erythematosus

Measuring lipid levels

When measuring lipids both the total cholesterol and HDL should be checked to provide the most accurate risk of CVD. A full lipid profile should also be checked (i.e. including triglycerides) before starting a statin. The samples do not need to be fasting.

In the vast majority of patients the cholesterol measurements will be fed into the QRISK2 tool. If however, the patient's cholesterol is very high we should consider familial hyperlipidaemia. NICE recommend the following that we should consider the possibility of familial hypercholesterolaemia and investigate further if:

- the total cholesterol level greater than 7.5 mmol/L and/or
- there is a personal or family history of premature coronary heart disease (an event before 60 years in an index person or first-degree relative [parents, siblings, children])

Interpreting the QRISK2 result

Probably the headline change in the 2014 guidelines was the new, lower cut-off of 10-year CVD risk cut-off of 10%.

NICE now recommend we offer a statin to people with a QRISK2 10-year risk of >= 10%

Lifestyle factors are of course important and NICE recommend that we give patients the option of having their CVD risk reassessed after a period of time before starting a statin.

Atorvastatin 20mg should be offered first-line.

Special situations

Type 1 diabetes mellitus

- NICE recommend that we 'consider statin treatment for the primary prevention of CVD in all adults with type 1 diabetes'
- atorvastatin 20 mg should be offered if type 1 diabetics who are:
    - older than 40 years, or
    - have had diabetes for more than 10 years or
    - have established nephropathy or
    - have other CVD risk factors

Chronic kidney disease (CKD)

- atorvastatin 20mg should be offered to patients with CKD
- increase the dose if a greater than 40% reduction in non-HDL cholesterol is not achieved and the eGFR > 30 ml/min. If the eGFR is < 30 ml/min a renal specialist should be consulted before increasing the dose

Secondary prevention

All patients with CVD should be taking a statin in the absence of any contraindication.

Atorvastatin 80mg should be offered first-line.

Follow-up of people started on statins

NICE recommend we follow up patients at 3 months

- repeat a full lipid profile
- if the non-HDL cholesterol has not fallen by at least 40% concordance and lifestyle changes should be discussed with the patient
- NICE recommend we consider increasing the dose of atorvastatin up to 80mg

Lifestyle modifications

These are in many ways predictable but NICE make a number of specific points:

Cardioprotective diet

- total fat intake should be <= 30% of total energy intake
- saturated fats should be <= 7% of total energy intake
- intake of dietary cholesterol should be < 300 mg/day
- saturated fats should be replaced by monounsaturated and polyunsaturated fats where possible
- replace saturated and monounsaturated fat intake with olive oil, rapeseed oil or spreads based on these oils
- choose wholegrain varieties of starchy food
- reduce their intake of sugar and food products containing refined sugars including fructose
- eat at least 5 portions of fruit and vegetables per day
- eat at least 2 portions of fish per week, including a portion of oily fish
- eat at least 4 to 5 portions of unsalted nuts, seeds and legumes per week

Physical activity

- each week aim for at least 150 minutes of moderate-intensity aerobic activity or 75 minutes of vigorous-intensity aerobic activity or a mix of moderate and vigorous aerobic activity

- do muscle-strengthening activities on 2 or more days a week that work all major muscle groups (legs, hips, back, abdomen, chest, shoulders and arms) in line with national guidance for the general population

Weight management

- no specific advice is given, overweight patients should be managed in keeping with relevant NICE guidance

Alcohol intake

- again no specific advice, other than the general recommendation: men and women are advised not to drink more than 14 units a week on a regular basis

Smoking cessation

- smokers should be encouraged to quit

+ Q 123

Next question

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 9 👎 16

Managing lipids in type 2 diabetes

NICE 👍 11 👎 14

2014 Lipid modification guidelines

Suggest link Report broken link

Score: **81.3%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✓
14	✓
15	✓
16	✓

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Question 17 of 342

A 45-year-old woman with a 2-year history of Graves' disease presents to the endocrine clinic with progressive ocular symptoms. Over the past month, she has noticed worsening proptosis associated with gritty sensations in the eyes, increased tearing, and episodes of diplopia, especially when looking upward. She is currently showing features of hyperthyroidism and is a smoker.

Examination reveals bilateral proptosis, conjunctival injection, and lid lag. Her endocrinologist is considering treatment options for her hyperthyroidism.

Which treatment option is most likely to worsen her symptoms?

○ Carbimazole ✕
○ Prednisolone ✕
○ Radioiodine therapy ✕
○ Smoking cessation ✕
○ Total thyroidectomy ✕

Submit answer

Reference ranges

Score: **81.3%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
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10	✗
11	✓
12	✓
13	✓
14	✓
15	✓
16	✓
17	-

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Question 17 of 342 ✓

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Examination reveals bilateral proptosis, conjunctival injection, and lid lag. Her endocrinologist is considering treatment options for her hyperthyroidism.

Which treatment option is most likely to worsen her symptoms?

Carbimazole	7%
Prednisolone	4%
[✓ Radioiodine therapy]	85%
Smoking cessation	4%
Total thyroidectomy	0%

If there is active Grave's eye disease then radioiodine therapy is not recommended as it can worsen the eye disease

Important for me Less important

Radioiodine therapy is the correct answer. Radioiodine can worsen thyroid eye disease (TED), particularly in patients who are active smokers or have active orbitopathy. The proposed mechanism involves increased exposure to thyroid autoantigens following tissue destruction, which amplifies the autoimmune response in the orbit. RAI should be avoided in patients with active TED unless necessary, and in such cases, high-dose corticosteroids (e.g., prednisolone) should be given as prophylaxis. In this patient, an active smoker with active TED, RAI carries a high risk of exacerbating the disease.

Carbimazole is a thionamide antithyroid medication used to restore and maintain euthyroidism. It is typically the first-line therapy for hyperthyroidism in patients with active TED. Maintaining an euthyroid state is essential, as both hyper- and hypothyroidism can worsen TED. Carbimazole is safe and does not increase the risk of orbital inflammation.

Prednisolone is a systemic corticosteroid used to treat moderate-to-severe active TED. It reduces orbital inflammation, alleviates symptoms, and may stabilise disease progression. When RAI is necessary, prednisolone is used prophylactically to mitigate the risk of TED worsening. It is not associated with harm in this setting and may offer protection.

Smoking cessation significantly reduces the risk of TED progression and enhances response to treatment. Smoking is the most important modifiable risk factor for TED, particularly in those with Graves' hyperthyroidism. Stopping smoking is a key intervention in disease management and should be encouraged in all patients.

Total thyroidectomy is a definitive treatment for hyperthyroidism and does not exacerbate TED. In fact, it may be preferred in patients with moderate-to-severe orbitopathy who are poor candidates for RAI. Unlike RAI, surgery avoids the immune flare caused by thyroid tissue destruction. It is a safe and effective alternative in active TED.

👍 👎 Discuss Improve

Next question

Thyroid eye disease ★

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology

- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention

- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features

- the patient may be eu-, hypo- or hyperthyroid at the time of presentation
- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management

- smoking cessation
- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery

Complications

Exposure keratopathy

- this is the most common complication of thyroid eye disease

- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy

- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia

- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)
- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):

- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes
- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy
Management of Graves' Orbitopathy

👍 9 👎 8

Suggest link

Report broken link

Score: **82.4%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓

10 ✗
11 ✓
12 ✓
13 ✓
14 ✓
15 ✓
16 ✓
17 ✓

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Question 18 of 342

A 54-year-old man attends with persistent hypertension. His blood pressure is 175/101mmHg.

His past medical history includes type 2 diabetes mellitus for which he takes metformin.

Tests are arranged and the results are detailed below:

Na^+	136 mmol/L	(135 - 145)
K^+	3.3 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	65 µmol/L	(55 - 120)

Aldosterone	680 pmol/L	(100-450)
Renin	0.3 mU/L	(5.4-30)

A saline infusion test is completed and the results post-infusion are as follows:

Aldosterone	550 pmol/L	(100-450)

The patient is keen to undergo surgical management of his condition if possible.

What investigation would be the most reliable in confirming the location of the lesion in this case?

- ○ Adrenal venous sampling ×
- ○ Delayed contrast-enhanced CT adrenals ×

○ Fine-needle aspiration adrenal biopsy ×

○ MRI adrenals ×

○ MRI brain ×

Submit answer

Reference ranges

Score: **82.4%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
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11	✓
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17	✓
18	-

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A saline infusion test is completed and the results post-infusion are as follows:

Aldosterone	550 pmol/L	(100-450)

The patient is keen to undergo surgical management of his condition if possible.

What investigation would be the most reliable in confirming the location of the lesion in this case?

Adrenal venous sampling	68%
Delayed contrast-enhanced CT adrenals	15%

Fine-needle aspiration adrenal biopsy	1%
MRI adrenals	16%
MRI brain	1%

Adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism

Important for me Less important

The most likely diagnosis here is hyperaldosteronism. Adrenal adenomas and adrenal hyperplasia are the most common causes. Secondary hyperaldosteronism can occur due to renin-angiotensin-aldosterone system overactivity as seen in cardiac failure, ascites, and fibromuscular dysplasia. In this case, renin is suppressed, so primary hyperaldosteronism is the most likely answer.

Adrenal venous sampling is the correct answer. This involves sampling blood from the adrenal veins and assessing the levels of aldosterone. This is the gold-standard test to distinguish between unilateral adrenal adenoma and bilateral hyperplasia and should be completed to confirm the location of the abnormality if surgery is to be considered. CT scanning can help identify lesions, however, many adrenal adenomas are non-functioning and not clinically relevant. Hormonal studies via AVS are therefore advised to confirm the location to avoid the removal of an adrenal gland unnecessarily.

Delayed contrast-enhanced CT adrenals is incorrect. Although CT scanning is a good modality to look for adrenal pathology many adrenal adenomas can be too small to pick up on CT. CT can also pick up incidental adenomas that are non-functioning and of no clinical consequence. CT is therefore not the most reliable modality to use to confirm the location of the lesion.

Fine-needle aspiration adrenal biopsy is incorrect. Adrenal adenomas are usually extremely small and not amenable to biopsy.

MRI adrenals is incorrect. Although MRI can be used, there are similar pitfalls to CT scanning as detailed above - MRI may pick up non-functioning adrenal adenomas or miss small adenomas.

MRI brain is incorrect. Pituitary lesions are not implicated in primary hyperaldosteronism.

👍 👎 Discuss (5) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes
- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

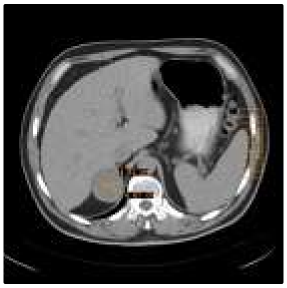
Features
- **hypertension**
  - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
  - e.g. muscle weakness
  - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
    - hypertension with hypokalemia
    - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
    - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
    - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

Suggest link

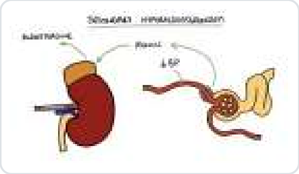
Report broken link

Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

Score: **77.8%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
9	✓
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14	✓
15	✓
16	✓
17	✓
18	✗

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A 43-year-old man presents to his general practitioner complaining of generalized aches and pains. He has a past medical history of glaucoma and takes acetazolamide. He does not smoke cigarettes or drink alcohol. He is a marketing manager for a large international fashion brand.

On examination, he has subtle proximal myopathy. There is no synovitis.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	2.8 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
Bicarbonate	16 mmol/L	(22-29)
CRP	4 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	165 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
Parathyroid hormone	7.9 pmol/L	(1.6-6.9)
Calcium	2.08 mmol/L	(2.20-2.60)
Vitamin D	31nmol/L	(>50)

What is the unifying diagnosis that explains the presentation?

○ Osteoporosis ×

○ Primary hyperparathyroidism ×

○ Type 1 renal tubular acidosis ×

○ Type 2 renal tubular acidosis ×

○ Type 4 renal tubular acidosis ×

Submit answer

Reference ranges

Score: **77.8%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
8	✗
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14	✓

15	✓
16	✓
17	✓
18	✗
19	-

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A 43-year-old man presents to his general practitioner complaining of generalized aches and pains. He has a past medical history of glaucoma and takes acetazolamide. He does not smoke cigarettes or drink alcohol. He is a marketing manager for a large international fashion brand.

On examination, he has subtle proximal myopathy. There is no synovitis.

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Bilirubin	14 µmol/L	(3 - 17)
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ALT	23 u/L	(3 - 40)
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Albumin	36 g/L	(35 - 50)
Parathyroid hormone	7.9 pmol/L	(1.6-6.9)
Calcium	2.08 mmol/L	(2.20-2.60)
Vitamin D	31nmol/L	(>50)

What is the unifying diagnosis that explains the presentation?

Osteoporosis	3%
Primary hyperparathyroidism	4%
Type 1 renal tubular acidosis	22%
[✓ Type 2 renal tubular acidosis]	68%
Type 4 renal tubular acidosis	3%

Hypokalaemia, osteomalacia - type 2 renal tubular acidosis

Important for me Less important

Type 2 renal tubular acidosis is the correct answer. Type 2 renal tubular acidosis is characterized by the failure of proximal tubular cells to reabsorb bicarbonate, resulting in academia. It is associated with hypokalemia and the development of osteomalacia. The traditional explanation for osteomalacia in this setting is that the proximal tubular conversion of 25 (OH)-cholecalciferol to the active 1,25 (OH)2-cholecalciferol is impaired. Acetazolamide treatment is a known cause of type 2 renal tubular acidosis. This patient presents with aches and pains and proximal myopathy, low vitamin D, low calcium and raised PTH, typical of osteomalacia. Additionally, there is academia (low serum bicarbonate) and hypokalemia. The unifying diagnosis is type 2 renal tubular acidosis secondary to acetazolamide treatment.

Type 4 renal tubular acidosis is incorrect. This is usually associated with hyperkalemia rather than hypokalemia.

Type 1 renal tubular acidosis is incorrect. This is less likely to cause osteomalacia and not associated with carbonic anhydrase inhibitor use. It is associated with the development of nephrocalcinosis.

Primary hyperparathyroidism is incorrect. This patient has an appropriately raised parathyroid hormone as a consequence of vitamin D deficiency and hypocalcemia.

Osteoporosis is incorrect. Calcium is typically normal in this condition and it is characterized by fracture and low T scores, rather than proximal myopathy and generalized aches and pains.

👍 👎 Discuss (4) Improve

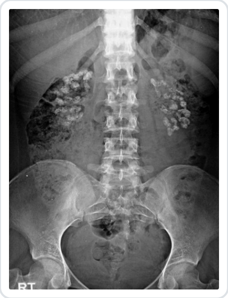
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube

👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **78.9%**

1	✓
2	✓
3	✓
4	✓
5	✗
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15	✓
16	✓
17	✓
18	✗
19	✓

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A 22 year old lady presents over a year with mild-moderate, intermittent abdominal pain. She has felt low in mood over this period and also her periods have stopped. Her history includes two previous attacks of renal calculi formation managed conservatively. She had a car crash recently, where she says that the car just 'came out of nowhere'. She is intermittently getting global headaches that can be very severe in nature but are otherwise featureless. On prompting, she tells you that she has sometimes noticed a white nipple discharge on her clothing. She has had low blood pressure and several faints over this last year and after her GP discovered a low serum cortisol level he has started her on oral hydrocortisone and referred her to your clinic. On examination today her blood pressure 130/80. She has a blistering, red rash across her lower abdomen and back. Her abdomen is largely non-tender with no palpable organomegally or peritonism. Visual fields are reduced bitemporally. Her urine dipstick shows glycosuria. The remainder of the examination is unremarkable. Which of the following is likely to treat the underlying condition most effectively?

- ○ Bisphosphonates ×
- ○ Surgery ×
- ○ Cabergoline ×
- ○ Octreotide ×
- ○ Insulin ×

Submit answer

Reference ranges

Score: **78.9%**

1	✓
2	✓
3	✓
4	✓
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13	✓
14	✓
15	✓
16	✓
17	✓
18	✗
19	✓
20	-

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Question 20 of 342 ✗

A 22 year old lady presents over a year with mild-moderate, intermittent abdominal pain. She has felt low in mood over this period and also her periods have stopped. Her history includes two previous attacks of renal calculi formation managed conservatively. She had a car crash recently, where she says that the car just 'came out of nowhere'. She is intermittently getting global headaches that can be very severe in nature but are otherwise featureless. On prompting, she tells you that she has sometimes noticed a white nipple discharge on her clothing. She has had low blood pressure and several faints over this last year and after her GP discovered a low serum cortisol level he has started her on oral hydrocortisone and referred her to your clinic. On examination today her blood pressure 130/80. She has a blistering, red rash across her lower abdomen and back. Her abdomen is largely non-tender with no palpable organomegally or peritonism. Visual fields are reduced bitemporally. Her urine dipstick shows glycosuria. The remainder of the examination is unremarkable. Which of the following is likely to treat the underlying condition most effectively?

	Bisphosphonates	1%
🟩	Surgery	50%
	Cabergoline	38%
🟥	Octreotide	11%
	Insulin	0%

The unifying diagnosis here is is multiple endocrine neoplasia (MEN) type 1. MEN1 consists of tumours of the parathyroid, pituitary, and pancreas.

This patient has symptoms of hypercalcaemia (abdominal groans, renal stones, and psychic moans of the 'bones, stones, groans, psychic moans'). This can be caused by a parathyroid tumour. She has symptoms suggestive of a non-functioning pituitary macro-adenoma causing hyperprolactinemia (amenorrhea and galactorrhoea) and hypopituitarism presenting as Addison's from suppressed ACTH. Dopamine is the inhibitor of prolactin in the pituitary, and comes from the hypothalamus. A nonfunctioning pituitary macro-adenoma compresses the pituitary stalk, interrupting dopamine flow to the pituitary, and therefore abolishing dopaminergic inhibition of prolactin, resulting in hyperprolactinaemia. At the same time, the macro-adenoma causes hypopituitarism through local pressure effects on the pituitary itself. 76% of pituitary tumours in MEN1 are prolactinomas, with the remainder being nonfunctioning adenomas. Prolactinomas are extremely sensitive to medical management with e.g. cabergoline or bromocriptine and even shrink in size subsequently. First line treatment for nonfunctioning adenomas however is surgical removal. The gylcosuria is suggestive of hyperglycaemia, which in the context of this MEN picture suggests a glucagonoma.

The answers try and trick you into looking to manage just one of the abnormalities here in an isolated fashion, e.g. targeting the pituitary with cabergoline, the pancreas with insulin, or the parathyroid with bisphosphonates. This may may happen if you do not recognise that this is MEN. However, as the question asks what treatment is likely to treat the underlying condition, clearly insulin alone, cabergoline alone, or bisphosphonate alone will not suffice. Of the options available, only surgery can tackle all of the problems, and indeed is what most patients with MEN end up needing.

MEN is an autosomal dominant condition. MEN1 consists of tumours of the parathyroid, anterior pituitary, and pancreas:

- The parathyroid tumours cause hypercalcaemia and its symptoms (which don't forget include polyuria and polydypsia). The management of hypercalcaemia is fluid resuscitation and bisphosphonates.

- Pituitary tumours can be a prolactinoma, somatotroph adenoma (causing acromegally by secreting growth hormone), or an ACTH-secreting tumour causing Cushing's disease. There can be any combination of these, although the question will usually point you toward one particular pituitary abnormality. Remember that you are unlikely to elicit a galactorrhoea history unless you specifically ask and are sensitive about it.

- The pancreas tumours can be an insulinoma (persistent hypoglycaemia -check C-peptide which will be high in endogenous insulin secretion as opposed to exogenously given insulin e.g. in self harm/non-accidental injury, where c-peptide levels remain low.), gastrinoma (Zollinger-Ellison, presenting as refractory gastric ulcers), glucagonoma causing persistent hyperglycaemia and also a necrolytic migratory erythema (as in this case), or a VIPoma causing profuse watery diarrhoea (VIP is the antagonist to gastrin and therefore tries to get you to flush the gut out rather than hold and digest contents.

MEN II has two forms. MEN2a presents with medullary thyroid carcinoma (neck lump), parathyroid tumour (hypercalcemia) and pheochromocytoma (hypertension, flushing, tachycardia intermittently). MEN2b presents with medullary thyroid carcinoma and pheochromocytoma.

The key message for the exam is that if you are presented with a case where you suspect there is an endocrine abnormality (even just a high calcium), look again at the stem to make sure you are not missing other endocrine abnormalities being present (e.g. amenorrhoea) that might alert you to there being an underlying diagnosis of MEN. If you know a few key features of each of the possible abnormalities within each type of MEN, you should be able to pinpoint which MEN type is present.

References:

- http://thejns.org/doi/abs/10.3171/foc.2004.16.4.3
- http://press.endocrine.org/doi/abs/10.1210/jcem.81.7.8675591
- http://www.ncbi.nlm.nih.gov/pubmed/10843153
- http://www.endocrine-abstracts.org/ea/0029/ea0029MTE16.htm

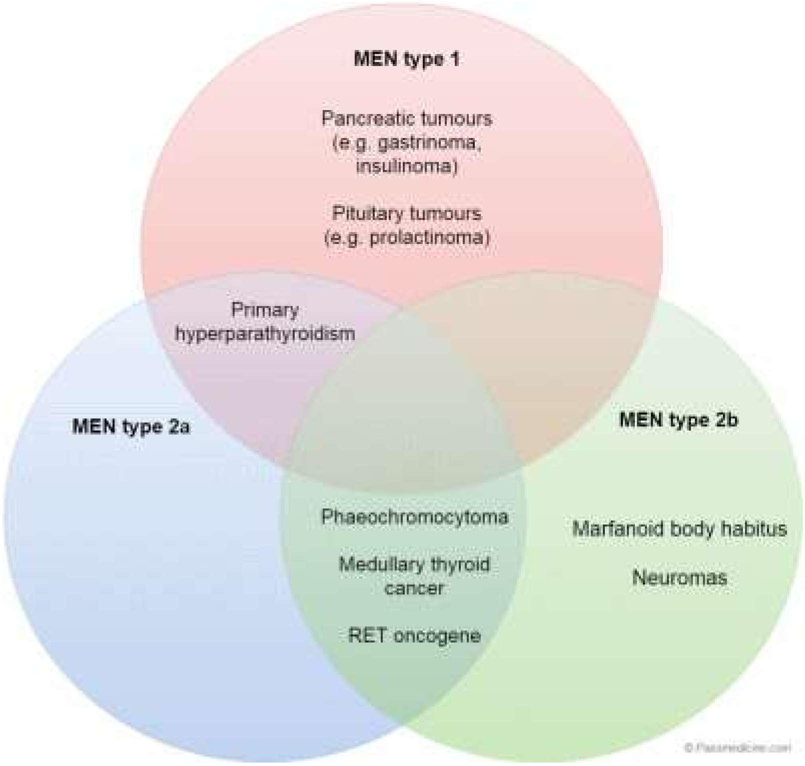
👍 👎 Discuss (15) Improve

Next question

Multiple endocrine neoplasia

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
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Venn diagram showing the different types of MEN and their associated features

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Pancreatic Neuroendocrine Neoplasms

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **75%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
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13	✓
14	✓
15	✓
16	✓
17	✓
18	✗
19	✓

20 ✕

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← Question 21 of 342 →

A 31-year-old woman is admitted with nausea and vomiting. She is 10 weeks into her first pregnancy. She reports vomiting multiple times daily and has been unable to tolerate oral anti-emetics or fluids for over 24 hours. You suspect hyperemesis gravidarum and commence intravenous fluids.

What treatment is recommended first-line?

- ○ Cyclizine ✕
- ○ Haloperidol ✕
- ○ Metoclopramide ✕
- ○ Ondansetron ✕
- ○ Pyridoxine ✕

Submit answer

Reference ranges

Score: **75%**

1 ✓
2 ✓
3 ✓
4 ✓
5 ✗

6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✓
14	✓
15	✓
16	✓
17	✓
18	✗
19	✓
20	✗
21	-

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Question 21 of 342 ✗

A 31-year-old woman is admitted with nausea and vomiting. She is 10 weeks into her first pregnancy. She reports vomiting multiple times daily and has been unable to tolerate oral anti-emetics or fluids for over 24 hours. You suspect hyperemesis gravidarum and commence intravenous fluids.

What treatment is recommended first-line?

Cyclizine	64%
Haloperidol	0%
Metoclopramide	9%
Ondansetron	15%
Pyridoxine	12%

Antihistamines are first-line in the management of nausea & vomiting in pregnancy/hyperemesis gravidarum

Important for me Less important

The correct answer is **cyclizine**. Anti-histamines such as cyclizine or promethazine are recommended first-line for hyperemesis gravidarum.

Haloperidol is incorrect. Haloperidol is an anti-psychotic but is associated with anti-emetic effects. However, it is not recommended in RCOG guidance. It has more of a role in managing nausea and vomiting in palliative care.

Metoclopramide is incorrect. This is a prokinetic agent with useful anti-emetic properties. It carries risks of extrapyramidal side effects such as dystonia so isn't recommended first-line or for long courses.

Ondansetron is incorrect. This is an option if first-line treatments are not effective or not tolerated. However, there is a slightly increased risk of cleft lip if used in the first trimester. For this reason, it requires specific counselling and should not be used first-line.

Pyridoxine is incorrect. Pyridoxine has been used in hyperemesis in some cases, often in combination with doxylamine, particularly outside the UK. However, it is not yet recommended in RCOG guidelines.

👍 👎 Discuss (2) Improve

Next question

Hyperemesis gravidarum

Whilst the majority of women experience nausea (previously termed 'morning sickness') during the early stages of pregnancy it can become problematic in a minority of cases. The Royal College of Obstetricians and Gynaecologists (RCOG) now use the term 'nausea and vomiting of pregnancy' (NVP) to describe troublesome symptoms, with hyperemesis gravidarum being the extreme form of this condition.

It occurs in around 1% of pregnancies and is thought to be related to raised beta hCG levels. Hyperemesis gravidarum is most common between 8 and 12 weeks but may persist up to 20 weeks.

Risk factors
- increased levels of beta-hCG
    - multiple pregnancies
    - trophoblastic disease
- nulliparity
- obesity
- family or personal history of NVP

Smoking is associated with a decreased incidence of hyperemesis.

Referral criteria for nausea and vomiting in pregnancy

NICE recommend considering admission in the following situations:
- Continued nausea and vomiting and is unable to keep down liquids or oral antiemetics
- Continued nausea and vomiting with ketonuria and/or weight loss (greater than 5% of body weight), despite treatment with oral antiemetics
- A confirmed or suspected comorbidity (for example she is unable to tolerate oral antibiotics for a urinary tract infection)

They also recommend having a lower threshold for admission to hospital if the woman has a co-existing condition (for example diabetes) that may be adversely affected by nausea and vomiting.

Hyperemesis gravidarum

The Royal College of Obstetricians and Gynaecologists (RCOG) recommend that the following triad is present before diagnosis of hyperemesis gravidarum: RCOG
- 5% pre-pregnancy weight loss
- dehydration
- electrolyte imbalance

Validated scoring systems such as the Pregnancy-Unique Quantification of Emesis (PUQE) score can be used to classify the severity of NVP.

Management
- simple measures
    - rest and avoid triggers e.g. odours
    - bland, plain food, particularly in the morning
    - ginger
    - P6 (wrist) acupressure
- first-line medications
    - antihistamines: oral cyclizine or promethazine

  - phenothiazines: oral prochlorperazine or chlorpromazine
  - combination drug doxylamine/pyridoxine: pyridoxine (vitamin B6) monotherapy is actually used commonly outside of the UK as a first-line treatment for NVP. However, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines
- second-line medications
  - oral ondansetron: ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate. The Medicines and Healthcare products Regulatory Agency (MHRA) advise that if ondansetron is used then these risks should be discussed with the pregnant woman
  - oral metoclopramide or domperidone: metoclopramide may cause extrapyramidal side effects. It should therefore not be used for more than 5 days
- admission may be needed for IV hydration
  - see criteria above for referral/admission
  - normal saline with added potassium is used to rehydrate

Women with hyperemesis gravidarum may develop dehydration, weight loss and electrolyte imbalances. Other complications include:

- acute kidney injury
- Wernicke's encephalopathy
- oesophagitis, Mallory-Weiss tear
- venous thromboembolism
- fetal outcome
  - studies generally show little evidence of adverse outcomes for birth weight/other markers for mild-moderate symptoms
  - severe NVP resulting in multiple admissions and failure to 'catch-up' weight gain may be linked to a small increase in preterm birth and low birth weight

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Obstetricians and Gynaecologists 👍 11 👎 13

2016 The Management of Nausea and Vomiting of Pregnancy and Hyperemesis Gravidarum

Clinical Knowledge Summaries 👍 8 👎 4

CKS - NICE vomiting in pregnancy guidance summary

Suggest link Report broken link

Score: **71.4%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
7	✓
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9	✓
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15	✓
16	✓
17	✓
18	✗
19	✓
20	✗
21	✗

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Question 22 of 342

A 75-year-old woman presents to her general practitioner (GP) due to increased tiredness and weight gain. Her past medical history includes hypothyroidism and hypertension, for which she takes levothyroxine and amlodipine.

She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

What is the most likely cause of her symptoms?

○ Bowel malignancy ×

○ Addison's disease ×

○ Amlodipine toxicity ×

○ Undercorrected hypothyroidism ×

○ Worsening anaemia ×

Submit answer

Reference ranges

Score: **71.4%**

1 ✓
2 ✓
3 ✓

4	✓
5	✗
6	✓
7	✓
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19	✓
20	✗
21	✗
22	-

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She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

What is the most likely cause of her symptoms?

Bowel malignancy	2%
Addison's disease	3%
Amlodipine toxicity	1%
🟩 Undercorrected hypothyroidism	91%
Worsening anaemia	3%

The most likely explanation here is undercorrected hypothyroidism. Ferrous sulphate is well known to reduce the absorption of other medications, particularly levothyroxine. Patients should be advised to take these tablets separately from their regular medications.

Although anaemia in a post-menopausal woman should always be investigated, there are no symptoms to suggest malignancy here. Addison's disease would be unlikely to cause weight gain.

👍 👎 Discuss (1) Improve

Next question

Hypothyroidism: causes ★

Hypothyroidism affects around 1-2% of women in the UK and is around 5-10 times more common in females than males.

Primary hypothyroidism

Hashimoto's thyroiditis
- most common cause
- autoimmune disease, associated with IDDM, Addison's or pernicious anaemia
- may cause transient thyrotoxicosis in the acute phase
- 5-10 times more common in women

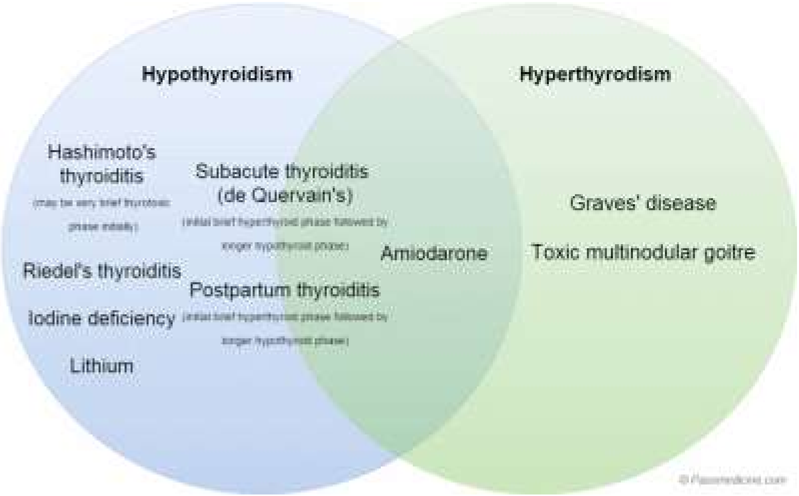
Subacute thyroiditis (de Quervain's)

Riedel thyroiditis

After thyroidectomy or radioiodine treatment

Drug therapy (e.g. lithium, amiodarone or anti-thyroid drugs such as carbimazole)

Dietary iodine deficiency



Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Secondary hypothyroidism (rare)

From pituitary failure

Other associated conditions
- Down's syndrome
- Turner's syndrome
- coeliac disease

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Clinical features

Thyroid disease

Podmedics - YouTube 👍 1 👎 0

HYPOTHYROIDISM
CAUSES

[Hypothyroidism and Hashimoto's Thyroiditis: Visual Explanation for Students]

Zero To Finals - YouTube 👍 2 👎 1

Report broken media

Score: **72.7%**

1	✓
2	✓
3	✓
4	✓
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16	✓
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An elderly male presents with a 2 week history of breathlessness. His past medical history includes diet-controlled type 2 diabetes, ischaemic heart disease, hypothyroidism and depression. His medication list includes levothyroxine, aspirin, simvastatin, ramipril, bisoprolol and citalopram. Observations on presentation to Emergency Department are as follows: respiratory rate 26/min, saturations 94% (on 4 litres oxygen via Venturi), heart rate 80 beats per minute, blood pressure 156/82 mmHg. Auscultation demonstrates crackles at the left base with no wheeze. The abdomen is soft and non-tender. There is no oedema peripherally.

Blood results on admission are provided below:

Hb	134 g/l
Platelets	172 * 10^9/l
WBC	13.3 * 10^9/l
Na^+	128 mmol/l
K^+	5.1 mmol/l
Urea	13 mmol/l
Creatinine	178 µmol/l
Serum osmolality	220 mosm/kg
Urinary sodium	50 mEq//l

What is the most likely cause of hyponatraemia?

○ Hypothyroidism

○ Chronic kidney disease

○ Addison's disease

○ Salt-losing nephropathy ✕

○ Syndrome of inappropriate antidiuretic hormone (SIADH) ✕

Submit answer

Reference ranges ⌄

Score: **72.7%**

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Question 23 of 342 ✓

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Creatinine	178 µmol/l
Serum osmolality	220 mosm/kg
Urinary sodium	50 mEq//l

What is the most likely cause of hyponatraemia?

Hypothyroidism	5%
Chronic kidney disease	8%
Addison's disease	11%

Salt-losing nephropathy	12%
Syndrome of inappropriate antidiuretic hormone (SIADH)	64%

This question demonstrates a common scenario in clinical practice. Management of hyponatraemia first requires clarification of fluid status (clinical hypovolaemia, euvolaemia or hypervolaemia), as differentials are influenced by this. This patient's history, examination findings and haemodynamic parameters are consistent with clinical euvolaemia.

Differentials for euvolaemic hyponatraemia would include hypothyroidism and SiADH. There are no clinical features suggestive of the former. Findings are consistent with community-acquired pneumonia with associated SiADH. This is confirmed by the presence of reduced serum osmolality and high urinary sodium. Measurement of urinary sodium concentration is an useful adjunct in helping to differentiate between hyponatraemia secondary to hypovolaemia and SiADH. With SiADH (and salt-wasting syndrome), the urinary sodium is high. With hypovolaemia, the urinary sodium is typically low.

👍 👎 💬 Discuss (15) Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology

- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water

balance

- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels
- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.
- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension, because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted, leading to hyponatraemia.

Causes of SIADH

Category	**Examples**
Malignancy	**small cell lung cancer** also: pancreas, prostate
Neurological	stroke subarachnoid haemorrhage subdural haemorrhage meningitis/encephalitis/abscess
Infections	tuberculosis pneumonia
Drugs	sulfonylureas* SSRIs, tricyclics carbamazepine vincristine cyclophosphamide
Other causes	positive end-expiratory pressure (PEEP)

Category	Examples
	• porphyrias

Investigations

- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.
- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management

- correction must be done slowly to avoid precipitating central pontine myelinolysis
- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

+ Q 123

Next question

Textbooks

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Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **73.9%**

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A 72-year-old Japanese female presents to the emergency department with sudden onset shortness of breath associated with palpitations. She has previously experienced similar palpitations 6 months ago but did not seek medical attention. She was last completely well and described by her daughter to be at baseline 24 hours ago when they had dinner together. The patient denies any chest pain, nausea, vomiting or sweating. On examination, the patient is pyrexic at 38.2 degrees and tachycardic, with a regular pulse at 130-140 beats per minute. Heart sounds demonstrate a gallop rhythm; auscultation of her chest reveals bibasal inspiratory coarse crackles and no wheeze. She has bilateral mild lower limb pitting oedema to low ankles. Examination of the abdominal and neurological systems is unremarkable. A chest radiograph demonstrates bibasal alveolar shadowing with mild bilateral pleural effusions. An ECG demonstrates sinus tachycardia at 130 beats per minute. Blood tests are as follows:

Hb	123 g/l
Platelets	299 * 10^9/l
WBC	9.5 * 10^9/l

Na^+	139 mmol/l
K^+	4.2 mmol/l
Urea	7.2 mmol/l
Creatinine	98 µmol/l

TSH	< 0.01 mU/l
Free T4	140 pmol/l
Free T3	40 pmol/l

Nursing staff have kindly taken blood cultures and taken measures to cool the patient. What is the next most appropriate immediate treatment?

○ Intravenous propranolol ×

○ Lugol's iodine ×

○ Oral carbimazole ×

○ Oral propylthiouracil ×

○ Oral prednisolone ×

Submit answer

Reference ranges ∨

Score: **73.9%**

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Urea	7.2 mmol/l
Creatinine	98 µmol/l

TSH	< 0.01 mU/l
Free T4	140 pmol/l
Free T3	40 pmol/l

Nursing staff have kindly taken blood cultures and taken measures to cool the patient. What is the next most appropriate immediate treatment?

Intravenous propranolol	78%
Lugol's iodine	6%
Oral carbimazole	3%
Oral propylthiouracil	10%
Oral prednisolone	4%

In thyroid storm with IV beta-blockers are a important first-line treatment

Important for me Less important

The patient has presented with sudden onset heart failure associated with sinus tachycardia, pyrexia and thyrotoxicosis: this represents a thyroid storm and is an endocrinological emergency. The treatment comprises of four aims: resuscitation, treat the sympathetic consequences of thyrotoxicosis, block underlying hyperthyroidism and treat any heart failure present. The first step involved intravenous followed by oral beta-blockade. Diltiazem is an appropriate alternative if the patient cannot tolerate beta blockers. In cases of simultaneous poor ventricular function and thyroid storm, intravenous infusions of short-acting beta blockers such as esmolol are also appropriate, which can be switched off immediately at the earliest sign of worsening cardiac function secondary to beta blockade. Thyroid blockers can be instituted after this immediate therapy. Oral corticosteroids are also important to reduce peripheral T4 to T3 conversion. However, both treatments can be instituted after achieving haemodynamic stability.

👍 👎 Discuss (12) Improve

Next question

Thyroid storm

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:
- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

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Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **75%**

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Question 25 of 342

You are asked to review a 42- year- old alcoholic who has been admitted to the medical ward following 48hrs of vomiting, generalised muscle weakness and palpitations. Despite two calcium infusions, the most recent measured calcium is still 1.89 mmol/l. On examination his blood pressure is 95/60 mmHg, pulse is 95 beats per minute and regular. You note intermittent runs of SVT on his cardiac monitor.

Other urea and electrolytes are shown below:

Na^+ 132	mmol/l
K^+ 3.7	mmol/l
Urea 5.4	mmol/l
Creatinine 82	μmol/l
Glucose 5.2	mmol/l

Which of the following is the most appropriate next step?

○ IV calcium ×

○ IV magnesium ×

○ IV potassium ×

○ IV phosphate ×

○ IV glucose ×

Submit answer

Reference ranges ⌵

Score: **75%**

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Na^+ 132	mmol/l
K^+ 3.7	mmol/l
Urea 5.4	mmol/l
Creatinine 82	μmol/l
Glucose 5.2	mmol/l

Which of the following is the most appropriate next step?

IV calcium	7%
IV magnesium	81%
IV potassium	3%
IV phosphate	8%
IV glucose	2%

Magnesium deficiency causes hypocalcaemia

Important for me Less important

Long term alcoholism as well as leading to falls in serum calcium, can also lead to significant falls in magnesium, which can account both for the persistently decreased calcium despite replacement, and for the runs of SVT seen here. The most appropriate next step is magnesium replacement, which is likely to facilitate both an improvement in calcium and resolution of SVT.

Further IV calcium will be ineffective without first replacing magnesium, and potassium is within the normal range, as is glucose. Phosphate replacement may be required in the treatment of alcoholism, but this is usually in the context of refeeding syndrome.

👍 👎 Discuss (3) Improve

Next question

Hypocalcaemia: causes and management

The clinical history combined with parathyroid hormone levels will reveal the cause of hypocalcaemia in the majority of cases

Causes
- vitamin D deficiency (osteomalacia)
- chronic kidney disease
- hypoparathyroidism (e.g. post thyroid/parathyroid surgery)
- pseudohypoparathyroidism (target cells insensitive to PTH)
- rhabdomyolysis (initial stages)
- magnesium deficiency (due to end organ PTH resistance)
- massive blood transfusion
- acute pancreatitis

Contamination of blood samples with EDTA may also give falsely low calcium levels.

Management
- severe hypocalcaemia (e.g. carpopedal spasm, tetany, seizures or prolonged QT interval) requires IV calcium replacement

  - the preferred method is with intravenous calcium gluconate, 10ml of 10% solution over 10 minutes
  - intravenous calcium chloride is more likely to cause local irritation
  - ECG monitoring is recommended
- further management depends on the underlying cause

+ Q 123

Next question

Textbooks

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Extended textbook

Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 3 👎 2

Report broken media

Score: **76%**

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Question 26 of 342

A 72-year-old male was admitted drowsy and confused. His family describe a 4-day history of shortness of breath and a productive cough. His past medical history includes type 2 diabetes mellitus, hypertension and hypercholesterolaemia. He usually takes metformin 500 mg three times daily, gliclazide 80 mg twice daily, amlodipine 5 mg daily and simvastatin 40 mg nightly. On examination he is confused with dry mucous membranes, blood pressure of 100/50 mmHg, a pulse of 110/min, a temperature of 37.6 ^{o}C and a respiratory rate of 20/min. Course crepitations were found at the right base and his pulse was thready with a capillary refill of 3 seconds; jugular venous pressure was not visible. Capillary blood glucose was found to be HI.

A venous blood sample is taken:

Hb	129 g/l	Na$^+$	161 mmol/l
Platelets	204 * 10^9/l	K$^+$	4.9 mmol/l
WBC	13.1 * 10^9/l	Urea	15.2 mmol/l
Neuts	11.9 * 10^9/l	Creatinine	97 µmol/l
Glucose	56 mmol/l	eGFR	62 mg/l
Ketones	1.9 mmol/l	HbA1c	75 mmol/mol
pH	7.35	HCO$_3$	20 mmol/mol

What treatment would you initiate first?

- ○ 0.9% normal saline ×
- ○ 0.45 % normal saline ×
- ○ Hartmann's solution ×
- ○ Intravenous insulin ×

○ 5% dextrose ×

Submit answer

Reference ranges ⌄

Score: **76%**

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- ○ 0.45 % normal saline
- ○ Hartmann's solution
- ○ Intravenous insulin

○ 5% dextrose ×

Submit answer

Reference ranges

Score: **76%**

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Glucose	56 mmol/l	eGFR	62 mg/l
Ketones	1.9 mmol/l	HbA1c	75 mmol/mol
pH	7.35	HCO_3	20 mmol/mol

What treatment would you initiate first?

0.9% normal saline	82%
0.45 % normal saline	12%
Hartmann's solution	2%
Intravenous insulin	1%

5% dextrose 3%

The underlying diagnosis is hyperosmolar hyperglycaemic state (HHS) precipitated by a lower respiratory tract infection. The diagnosis is made when a patient shows marked hyperglycaemia and hypovolaemia with a serum osmolality > 320 mosm/kg in the absence of marked ketonaemia or acidosis. This patient is clinically dry with a glucose of 56 and a calculated osmolality of 2(161+4.9) + 56 + 15 = 403 mosm/kg with mild ketonaemia.

Despite this gentlemen's hypernatraemia, the first line fluid therapy is 0.9% normal saline as patients in HHS are sodium deplete. One litre should be administered rapidly and electrolytes then repeated to gauge potassium requirements in further fluid bags. A small initial rise in sodium is expected and this should not discourage further use of normal saline. Insulin is not routinely started immediately in the absence of significant ketonaemia as glucose will fall with fluid therapy alone. When glucose stops falling insulin may be started if glucose still remains high. Too rapid correction of osmolality i.e. with aggressive insulin and fluids in combination can precipitate cerebral oedema.

Guidelines for the management of HHS can be found below:
http://www.diabetologists-abcd.org.uk/JBDS/JBDSIPHHSAdults.pdf

👍 👎 Discuss (6) Improve

Next question

Hyperosmolar hyperglycaemic state ★

Hyperosmolar hyperglycaemic state (HHS) is a medical emergency that can be difficult to manage and has a significant associated mortality (up to 20%). Hyperglycaemia results in osmotic diuresis, severe dehydration, and electrolyte deficiencies. HHS typically presents in the elderly with type 2 diabetes mellitus (T2DM).

Pathophysiology

- hyperglycaemia → ↑ serum osmolality → osmotic diuresis → severe volume depletion

Precipitating factors

- intercurrent illness
- dementia
- sedative drugs

Clinical features

- whilst DKA presents within hours of onset, HHS comes on over many days, and consequently, the dehydration and metabolic disturbances may be more extreme
- consequences of volume loss
  - clinical signs of dehydration
  - polyuria
  - polydipsia
- systemic
  - lethargy
  - nausea and vomiting
- neurological
  - altered level of consciousness
  - focal neurological deficits
- haematological
  - hyperviscosity (may result in myocardial infarctions, stroke and peripheral arterial thrombosis)

There are no precise **diagnostic criteria** but the following are typically seen

- hypovolaemia
- marked hyperglycaemia (>30 mmol/L)
- significantly raised serum osmolarity (> 320 mosmol/kg)
  - can be calculated by: 2 * Na^+ + glucose + urea
- no significant hyperketonaemia (<3 mmol/L)
- no significant acidosis (bicarbonate > 15 mmol/l or pH > 7.3 - acidosis can occur due to lactic acidosis or renal impairment)

Management

- fluid replacement

    - fluid losses in HHS are estimated to be between 100 - 220 ml/kg
    - IV 0.9% sodium chloride solution
    - typically given at 0.5 - 1 L/hour depending on clinical assessment
    - potassium levels should be monitored and added to fluids depending on the level
- insulin
    - should not be given unless blood glucose stops falling while giving IV fluids
- venous thromboembolism prophylaxis
    - patients are at risk of thrombosis due to hyperviscosity

Complications

- vascular complications may occur due to hyperviscosity:
    - such as myocardial infarction
    - stroke

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Diabetes Societies 👍 20 👎 13

The management of the hyperosmolar hyperglycaemic state (HHS)

Suggest link Report broken link

Score: **76.9%**

1	✓
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25 ✓

26 ✓

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A 19-year-old woman with a history of type 1 diabetes is brought to the Emergency department with nausea and vomiting. there is no history of diarrhoea. She also has coeliac disease. She follows a gluten free diet and takes a basal bolus insulin regime with a usual HbA1c of 53 mmol/mol. On examination her blood pressure is 100/80 mmHg with a postural drop of 20 mmHg. Pulse is 88 beats per minute and regular. She looks dehydrated and tanned, she puts her tan down to weeks in the garden after her exams.

Investigations

Hb	102 g/l	Na$^+$	129 mmol/l
Platelets	189 * 10^9/l	K$^+$	5.0 mmol/l
WBC	10.9 * 10^9/l	Urea	9.9 mmol/l
Neuts	6.2 * 10^9/l	Creatinine	113 µmol/l
Lymphs	1.1 * 10^9/l	CRP	42 mg/l
Eosin	1.5 * 10^9/l		

Which of the following is the most important intervention with respect to her management?

- ○ Fluid restriction ×
- ○ IV anti-emetic ×
- ○ IV hydrocortisone ×
- ○ IV normal saline ×
- ○ NG feeding ×

Submit answer

Reference ranges

Score: **76.9%**

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27	-

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Question 27 of 342 ✓

A 19-year-old woman with a history of type 1 diabetes is brought to the Emergency department with nausea and vomiting. there is no history of diarrhoea. She also has coeliac disease. She follows a gluten free diet and takes a basal bolus insulin regime with a usual HbA1c of 53 mmol/mol. On examination her blood pressure is 100/80 mmHg with a postural drop of 20 mmHg. Pulse is 88 beats per minute and regular. She looks dehydrated and tanned, she puts her tan down to weeks in the garden after her exams.

Investigations

Hb	102 g/l	Na^+	129 mmol/l
Platelets	189 * 10^9/l	K^+	5.0 mmol/l
WBC	10.9 * 10^9/l	Urea	9.9 mmol/l
Neuts	6.2 * 10^9/l	Creatinine	113 µmol/l
Lymphs	1.1 * 10^9/l	CRP	42 mg/l
Eosin	1.5 * 10^9/l		

Which of the following is the most important intervention with respect to her management?

Fluid restriction	1%
IV anti-emetic	0%
[✓ IV hydrocortisone]	85%
IV normal saline	13%
NG feeding	0%

In a patient with type 1 diabetes, such tight glycaemic control with hypoglycaemia would be considered very unusual. Coupled with the easy tanning, nausea, vomiting, and postural drop in blood pressure, this is highly suggestive of possible Addison's disease. The slight rise in eosinophil count, anaemia, hyponatraemia and potassium at the upper end of the normal range, all support the diagnosis.

In this situation corticosteroid replacement is crucial, without it normal saline replacement alone won't correct any hypotension, nor will it improve hyponatraemia. Although anti-emetics and NG feeding may be useful in restoring this patient to health, they won't correct underlying adrenal insufficiency.

👍 👎 Discuss (8) Improve

Next question

Addison's disease

Autoimmune destruction of the adrenal glands is the most common cause of primary hypoadrenalism in the UK, accounting for 80% of cases. This is termed Addison's disease and results in reduced cortisol and aldosterone being produced.

Features
- lethargy, weakness, anorexia, nausea & vomiting, weight loss, 'salt-craving'
- hyperpigmentation (especially palmar creases)
    - ACTH is derived from a larger precursor molecule called proopiomelanocortin (POMC). When POMC is cleaved to produce ACTH, other melanocyte-stimulating hormones (MSH) are also produced. These MSHs have the effect of stimulating melanocytes in the skin to produce more melanin, the pigment responsible for skin colour
    - primary Addison's is associated with hyperpigmentation whereas secondary adrenal insufficiency is not
- vitiligo
- loss of pubic hair in women
- hypotension

- hypoglycaemia
- hyponatraemia and hyperkalaemia may be seen
- crisis: collapse, shock, pyrexia

Other causes of hypoadrenalism

Primary causes
- tuberculosis
- metastases (e.g. bronchial carcinoma)
- meningococcal septicaemia (Waterhouse-Friderichsen syndrome)
- HIV
- antiphospholipid syndrome

Secondary causes
- pituitary disorders (e.g. tumours, irradiation, infiltration)

Exogenous glucocorticoid therapy

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 10 👎 16

2017 Adrenal insufficiency â€“ recognition and management

Clinical Knowledge Summaries 👍 12 👎 9

Addison's disease

Suggest link Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 3 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **77.8%**

1 ✓
2 ✓
3 ✓

4	✓
5	✗
6	✓
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22	✓
23	✓
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26	✓
27	✓

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Question 28 of 342

You are asked to review an 82-year-old man on the care of the elderly ward as serum sodium results are low. The patient was admitted five hours ago with a diagnosis of community-acquired pneumonia and exacerbation of congestive cardiac failure. He presented with a productive cough, shortness of breath and leg swelling. He has a past medical history of multiple myocardial infarcts four years ago and has subsequently developed heart failure. He also has had osteoarthritis, hypertension and high cholesterol. He takes aspirin, ramipril, furosemide, bisoprolol, omeprazole and atorvastatin. On examination, he has a raised JVP and bilateral crepitations to midzones bilaterally as well as oedema to the sacrum.

Blood tests:

Hb	122 g/l
Platelets	478 * 10^9/l
WBC	13.9 * 10^9/l
Na^+	125 mmol/l
K^+	3.4 mmol/l
Urea	6.1 mmol/l
Creatinine	82 µmol/l

Urinary sodium: 15mmol/l

What is the most likely cause of hyponatraemia?

○ SIADH ×

○ Furosemide ×

○ Ramipril ×

○ Heart failure ×

○ Renal failure ×

Submit answer

Reference ranges ⌵

Score: **77.8%**

1	✓
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21	✗
22	✓
23	✓

24	✓
25	✓
26	✓
27	✓
28	-

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WBC	13.9 * 10^9/l
Na^+	125 mmol/l
K^+	3.4 mmol/l
Urea	6.1 mmol/l
Creatinine	82 μmol/l

Urinary sodium: 15mmol/l

What is the most likely cause of hyponatraemia?

SIADH	13%
Furosemide	10%
Ramipril	1%
Heart failure	75%

Renal failure 1%

In hyponatraemia, a low urinary sodium makes diuretics an unlikely cause

Important for me Less important

The most likely cause of this patient's hyponatraemia is heart failure. The patient is clinically hypervolaemic with hyponatraemia in the context of low urinary sodium. The low urinary sodium excludes diuretics as a cause of hyponatraemia. SIADH is also unlikely with low urinary sodium, and also cannot be diagnosed with hypervolaemia. Renal failure can cause hyponatraemia with low urinary sodium as the kidneys fail to excrete sodium, but the normal creatinine excludes this.

👍 👎 Discuss (4) Improve

Next question

Hyponatraemia ★

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)

- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic

- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss

- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

+ Q 123

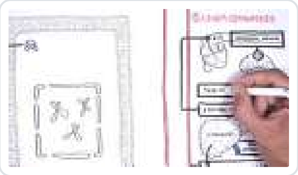
Next question

Textbooks

High-yield textbook

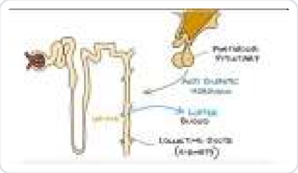
Extended textbook

Media



Hyponatraemia - classification, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 5 👎 0



Understanding Hyponatraemia

Zero To Finals - YouTube 👍 17 👎 1

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **75%**

1	✓
2	✓
3	✓
4	✓
5	✗
6	✓
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Question 29 of 342

A 45-year-old man with headaches returns to the endocrinology clinic following investigations. He originally complained of headaches waking him up at night, low energy, weight loss, low libido and postural dizziness. Investigations have revealed a normal full blood count, low free T4, low testosterone, low morning cortisol, low LH and FSH and low TSH. He also had an MRI which is suspicious for pituitary adenoma. He is keen to start whichever is necessary to feel better. He has no other medical problems and takes no regular tablets. Which hormonal replacement would be contra-indicated in the immediate setting?

- ○ Hydrocortisone
- ○ Prednisolone
- ○ Dexamethasone
- ● Testosterone
- ○ Levothyroxine

Submit answer

Reference ranges

Score: **75%**

1 ✓
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29	-

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	Hydrocortisone	6%
	Prednisolone	5%
	Dexamethasone	10%
🟥	Testosterone	24%
🟩	Levothyroxine	54%

In hypothyroidism with adrenal failure do not start thyroid hormone replacement before glucocorticoid replacement as otherwise an adrenal crisis can be precipitated

Important for me Less important

This patient has panhypopituitarism secondary to a pituitary adenoma. It is critical for patients with adrenal and thyroid insufficiency to replace steroid function prior to replacing thyroid function as otherwise an adrenal crisis can be precipitated. Therefore levothyroxine is contra-indicated in the immediate setting, even though the patient will need replacement after he has started steroid replacement. Typically he would need hydrocortisone split into three doses to replace his steroid needs, and this is preferred over dexamethasone and prednisolone.

Ultimately the treatment may be trans-sphenoidal surgery.

👍 👎 💬 Discuss (3) Improve

Next question

Hypothyroidism: levothyroxine therapy ★

Key points

- initial starting dose of levothyroxine should be lower in elderly patients and those with ischaemic heart disease. The BNF recommends that for patients with cardiac disease, severe hypothyroidism or patients over 50 years the initial starting dose should be 25mcg od with dose slowly titrated. Other patients should be started on a dose of 50-100mcg od
- following a change in thyroxine dose thyroid function tests should be checked after 8-12 weeks
- the therapeutic goal is 'normalisation' of the thyroid stimulating hormone (TSH) level. As the majority of unaffected people have a TSH value 0.5-2.5 mU/l it is now thought preferable to aim for a TSH in this range
- women with established hypothyroidism who become pregnant should have their dose increased by at least 25-50 micrograms levothyroxine due to the increased demands of pregnancy. The TSH should be monitored carefully, aiming for a low-normal value
- there is no evidence to support combination therapy with levothyroxine and liothyronine

Side-effects of thyroxine therapy

- hyperthyroidism: due to over treatment
- reduced bone mineral density
- worsening of angina
- atrial fibrillation

Interactions

- iron, calcium carbonate

  - absorption of levothyroxine reduced, give at least 4 hours apart

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

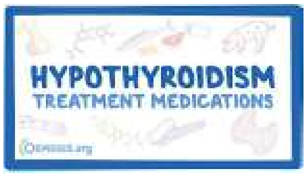
Links

Clinical Knowledge Summaries 👍 2 👎 1

Management of hypothyroidism

Suggest link Report broken link

Media


HYPOTHYROIDISM
TREATMENT MEDICATIONS
osmosis.org

Hypothyroidism treatment medications

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **72.4%**

1	✓
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22	✓
23	✓
24	✓
25	✓
26	✓
27	✓
28	✗
29	✗

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A 9-year-old boy is referred to the department of paediatrics by his general practitioner. He has developed secondary sexual characteristics at the age of 7. He has no significant past medical history and does not take any regular medications. His father commenced puberty at 9 years of age.

On examination, he has a coarse voice and facial hair. His testicles have enlarged. There is acne and adult body odour. The neurological examination is unremarkable. His blood pressure was 155/88 mmHg. There is no rash.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	388 * 10^9/L	(150 - 400)
WBC	4.2 * 10^9/L	(4.0 - 11.0)
Na$^+$	138 mmol/L	(135 - 145)
K$^+$	2.9 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Testosterone	42 ng/dl	(7-20)
FSH	1.2 IU/L	(<3)
LH	1.1 IU/L	(0.02-4.8)
TSH	1.2 mIU/L	(0.5-5.5)

From the listed options, which is the most likely diagnosis?

○ Brain tumour ✕

○ Hypothyroidism ×

○ Liddle's syndrome ×

○ McCune-Albright syndrome ×

○ 11-beta hydroxylase deficiency ×

Submit answer

Reference ranges ∨

Score: **72.4%**

1 ✓
2 ✓
3 ✓
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28	✗
29	✗
30	-

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← Question 30 of 342 ✗ →

A 9-year-old boy is referred to the department of paediatrics by his general practitioner. He has developed secondary sexual characteristics at the age of 7. He has no significant past medical history and does not take any regular medications. His father commenced puberty at 9 years of age.

On examination, he has a coarse voice and facial hair. His testicles have enlarged. There is acne and adult body odour. The neurological examination is unremarkable. His blood pressure was 155/88 mmHg. There is no rash.

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CRP	4 mg/L	(< 5)
Testosterone	42 ng/dl	(7-20)
FSH	1.2 IU/L	(<3)
LH	1.1 IU/L	(0.02-4.8)
TSH	1.2 mIU/L	(0.5-5.5)

From the listed options, which is the most likely diagnosis?

Brain tumour 1%

	Hypothyroidism	1%
	Liddle's syndrome	11%
🟥	McCune-Albright syndrome	20%
🟩	11-beta hydroxylase deficiency	68%

CAH due to 11-beta hydroxylase deficiency can cause apparent mineralocorticoid excess syndrome (AMES) resulting in hypertension and hypokalemia

Important for me Less important

11-beta hydroxylase deficiency is correct. This is the second most common cause of congenital adrenal hyperplasia. While CAH typically presents in the neonatal period, non-classical cases may present later with evidence of precocious puberty, such as in this case. Precocious puberty is when puberty occurs before 9 in males. This patient also has evidence of hypokalemia and hypertension, which can occur in this condition. The elevated serum testosterone but normal LH and FSH suggest a peripheral cause of precocious puberty (in this case adrenal) rather than a central cause.

A brain tumour is incorrect. The LH and FSH are normal. We would expect them to be raised with a central cause of precocious puberty.

Hypothyroidism is incorrect. While this can cause precocious puberty, the TSH is within normal limits.

McCune-Albright syndrome is incorrect. This is a cause of precocious puberty but typically it is associated with cafe-au-lait spots, which are absent here.

Liddle's syndrome is incorrect. This is an inherited renal tubular disorder that may cause hypertension and hypokalemia but it does not cause precocious puberty.

👍 👎 Discuss (4) Improve

Next question

Congenital adrenal hyperplasia: features

21-hydroxylase deficiency features

- virilisation of female genitalia
- precocious puberty in males
- 60-70% of patients have a salt-losing crisis at 1-3 wks of age

11-beta hydroxylase deficiency features

- virilisation of female genitalia
- precocious puberty in males
- hypertension
- hypokalaemia

17-hydroxylase deficiency features

- non-virilising in females
- inter-sex in boys
- hypertension

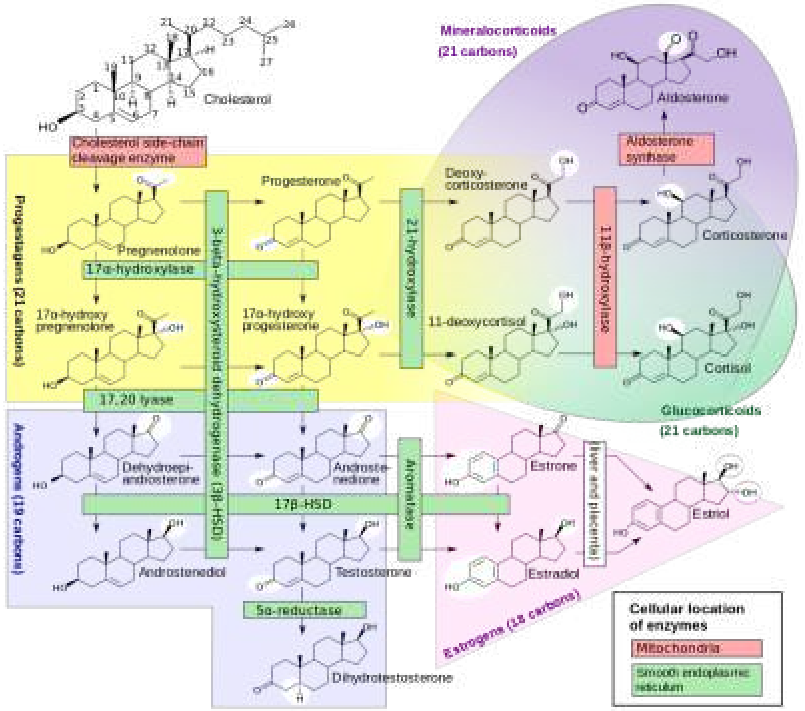

Cholesterol
Mineralocorticoids (21 carbons)
Aldosterone
Aldosterone synthase
Cholesterol side-chain cleavage enzyme
Progesterone
Deoxy-corticosterone
Corticosterone
Progestagens (21 carbons)
Pregnenolone
17α-hydroxylase
3-beta-hydroxysteroid dehydrogenase (3β-HSD)
21-hydroxylase
11β-hydroxylase
17α-hydroxy pregnenolone
17α-hydroxy progesterone
11-deoxycortisol
Cortisol
17,20 lyase
Glucocorticoids (21 carbons)
Androgens (19 carbons)
Dehydroepi-androsterone
Androste-nedione
Aromatase
Estrone
liver and placenta
Estriol
17β-HSD
Androstenediol
Testosterone
Estradiol
5α-reductase
Dihydrotestosterone
Estrogens (18 carbons)
Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

+ Q 123

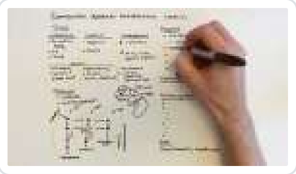
Next question

Textbooks

High-yield textbook

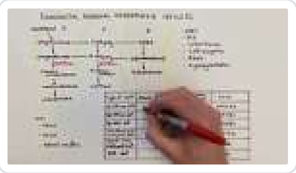
Extended textbook

Media



Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 👍 13 👎 0



Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 👍 10 👎 0

Report broken media

Score: **70%**

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30	✗

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A 36-year-old woman is referred to the hypertension clinic with difficult to control hypertension. She describes having occasional aches and pains which mainly affect their arms and legs. They have also noticed an increase in their urinary frequency and urgency. Her blood pressure reading is taken at the clinic and reads as 175/95 mmHg.

They are currently being treated with amlodipine and lisinopril for hypertension.

Bloods tests taken prior to attending the clinic are shown below.

Na^+	136 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

An aldosterone and renin test is performed and demonstrates an increased ratio of aldosterone to renin. Based on this, a CT abdomen is arranged. This is reported as showing bilateral adrenal hyperplasia.

What is the most appropriate management of this?

- ○ Bilateral adrenalectomy ×
- ○ Unilateral adrenalectomy ×
- ○ Watch and wait ×
- ○ Low sodium diet ×
- ○ Spironolactone ×

Submit answer

Reference ranges ∨

Score: **70%**

1	✓
2	✓
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23	✓
24	✓
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30	✗
31	-

Question 31 of 342 ✓

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Creatinine	110 µmol/L	(55 - 120)

An aldosterone and renin test is performed and demonstrates an increased ratio of aldosterone to renin. Based on this, a CT abdomen is arranged. This is reported as showing bilateral adrenal hyperplasia.

What is the most appropriate management of this?

Bilateral adrenalectomy	8%
Unilateral adrenalectomy	1%
Watch and wait	2%
Low sodium diet	2%
Spironolactone	88%

Primary hyperaldosteronism: manage with spironolactone

Important for me Less important

The correct answer is spironolactone. Bilateral adrenal hyperplasia can be safely and successfully managed with an aldosterone antagonist (either spironolactone or eplerenone) as this will reduce the effects of aldosterone.

There is no indication for surgical management in the treatment of bilateral adrenal hyperplasia so there is no rationale for an adrenalectomy. A low sodium diet is a reasonable lifestyle modification for hypertensive patients, but there is an underlying cause so medical management is more appropriate in this case.

Watch and wait would be inappropriate as there is a clear diagnosis and a treatment.

👍 👎 Discuss (6) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes
- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

Features

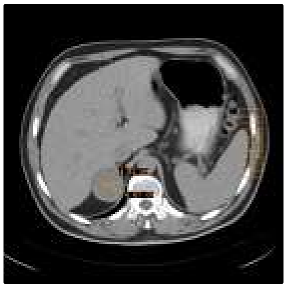
- **hypertension**
    - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
    - e.g. muscle weakness
    - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
    - hypertension with hypokalemia
    - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
    - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
    - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

B I

Textbooks

High-yield textbook

Extended textbook

Links

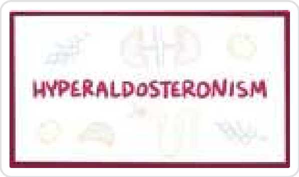
The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

Suggest link

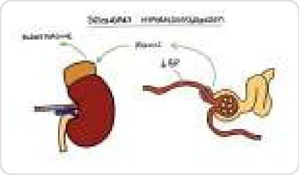
Report broken link

Media



Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

Score: **71%**

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Question 32 of 342

An 85-year-old woman is brought into A+E with hypothermia, sinus bradycardia and unresponsive. A CT head reveals no acute intra-cranial pathology. Passive warming and intravenous fluids are commenced. Subsequent blood tests reveal the panel below. A collateral history from family members reveals symptoms of lethargy, cold intolerance and weight gain over the last few months.

TSH	>30.0 mU/L
T3	<0.05 mU/L

What is the most appropriate initial treatment?

- ○ Levothyroxine and Liothyronine ✕
- ○ Levothyroxine ✕
- ○ Lugol's iodine ✕
- ○ Liothyronine ✕
- ○ Carbimazole ✕

Submit answer

Reference ranges

Score: **71%**

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32	-

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Back to top

An 85-year-old woman is brought into A+E with hypothermia, sinus bradycardia and unresponsive. A CT head reveals no acute intra-cranial pathology. Passive warming and intravenous fluids are commenced. Subsequent blood tests reveal the panel below. A collateral history from family members reveals symptoms of lethargy, cold intolerance and weight gain over the last few months.

TSH	>30.0 mU/L
T3	<0.05 mU/L

What is the most appropriate initial treatment?

🟩	Levothyroxine and Liothyronine	40%
🟥	Levothyroxine	40%
	Lugol's iodine	6%
	Liothyronine	12%
	Carbimazole	2%

This is myxedema coma, which is rare but has a high mortality rate. Administration of both liothyronine (T3) and levothyroxine (T4) is the most appropriate treatment. Liothyronine (T3) has a greater biologic activity and faster onset than levothyroxine (T4) and should be continued until there is clinical improvement. The administration of hydrocortisone is also important as patients there may be coexisting adrenal insufficiency (primary or secondary).
Supportive measures are important such as re-warming, fluid resuscitation, electrolyte correction and vasopressors if required.

Carbimazole is used for hyperthyroidism.
Lugols iodine is used for rapid control of hyperthyroidism such as a

thyroid storm or prior to a total thyroidectomy for poor medical compliance.

Hypothyroidism: levothyroxine therapy ★

Key points
- initial starting dose of levothyroxine should be lower in elderly patients and those with ischaemic heart disease. The BNF recommends that for patients with cardiac disease, severe hypothyroidism or patients over 50 years the initial starting dose should be 25mcg od with dose slowly titrated. Other patients should be started on a dose of 50-100mcg od
- following a change in thyroxine dose thyroid function tests should be checked after 8-12 weeks
- the therapeutic goal is 'normalisation' of the thyroid stimulating hormone (TSH) level. As the majority of unaffected people have a TSH value 0.5-2.5 mU/l it is now thought preferable to aim for a TSH in this range
- women with established hypothyroidism who become pregnant should have their dose increased by at least 25-50 micrograms levothyroxine due to the increased demands of pregnancy. The TSH should be monitored carefully, aiming for a low-normal value
- there is no evidence to support combination therapy with levothyroxine and liothyronine

Side-effects of thyroxine therapy
- hyperthyroidism: due to over treatment
- reduced bone mineral density
- worsening of angina
- atrial fibrillation

Interactions
- iron, calcium carbonate

    - absorption of levothyroxine reduced, give at least 4 hours apart

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

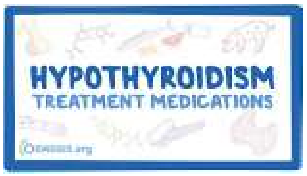
Clinical Knowledge Summaries 👍 2 👎 1

Management of hypothyroidism

Suggest link

Report broken link

Media


HYPOTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hypothyroidism treatment medications

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **68.8%**

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31 ✓

32 ✗

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A 31-year-old female presents with a 2-day history of abdominal pain, a 5-day history of diarrhoea and vomiting and reduced appetite. She is a known type one diabetic with background diabetic retinopathy and stage 3 chronic kidney disease. She usually takes 32 units lantus at night and variable doses of Novomix with meals however due to her poor appetite she has not taken these for 2 days. On examination, she looks unwell. The airway is patent and chest is clear. Respiratory rate is 26/min with normal oxygen saturations on air. Pulse is 120/min and thready with a capillary refill of 3 seconds centrally. Blood pressure is 103/45 mmHg with a temperature of 36.7°C. Abdomen is generally tender without guarding. Capillary blood glucose is 26 and ketones are 4.9. Arterial blood gas is as follows:

pH	7.32
pO2	11.6 kPa
pCO2	3.32 kPa
Bicarbonate	14 mmol/l
Base Excess	-6.5 mmol/l
Lactate	2.1 mmol/l

The patient is currently being fluid resuscitated appropriately. What form of insulin therapy would you advise?

○ Dose of novomix now and increase usual lantus by 8 units ×

○ Variable rate i.v insulin, continue all s/c insulins ×

○ Recommence normal s/c insulin regimen ×

○ Fixed rate i.v insulin, continue lantus ×

○ Fixed rate i.v insulin, stop all s/c insulin ×

Submit answer

Reference ranges ⌄

Score: **68.8%**

1	✓
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pO2	11.6 kPa
pCO2	3.32 kPa
Bicarbonate	14 mmol/l
Base Excess	-6.5 mmol/l
Lactate	2.1 mmol/l

The patient is currently being fluid resuscitated appropriately. What form of insulin therapy would you advise?

Dose of novomix now and increase usual lantus by 8 units	1%
Variable rate i.v insulin, continue all s/c insulins	4%
Recommence normal s/c insulin regimen	1%
[✓ Fixed rate i.v insulin, continue lantus]	75%
Fixed rate i.v insulin, stop all s/c insulin	19%

This patient has a compensated metabolic acidosis and despite her reasonably preserved pH, a bicarbonate of 14 suggests significant metabolic acidosis. In the presence of high blood ketones and hyperglycaemia, the diagnosis is diabetic ketoacidosis (DKA). Fluid therapy and potassium supplementation is a key part of DKA management but will not be dealt with in this question. Current recommendations advise fixed rate insulin with the addition of 10 % dextrose when capillary blood glucose drops below 14. The aim is to correct the underlying ketoacidosis brought about by a hypoinsulinaemic state. Patients should be continued on their long acting insulin but short/medium term insulins should be held. This ensures a smooth transfer from fixed rate to usual insulin regimen when the patient has clinically and biochemically improved.

Guidelines for the management of DKA can be found here:
https://www.bsped.org.uk/clinical/docs/DKAManagementOfDKAinAdultsMarch20101.pd

👍 👎 Discuss (3) Improve

Next question

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed

insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management
- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low

    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil

Potassium level in first 24 hours (mmol/L)	**Potassium replacement in mmol/L of infusion solution**
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to

monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

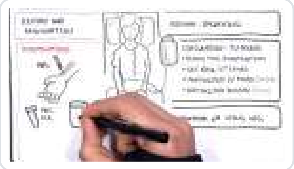
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

Report broken link

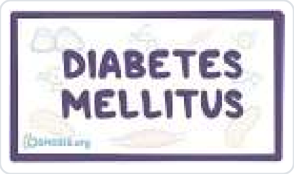
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2


DIABETES
MELLITUS
osmosis.org

Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **69.7%**

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A 33-year-old female is brought into the emergency department as a stand-by. She has felt unwell for the past 2 weeks. She describes lethargy, light-headedness and occasional shortness of breath. More recently she developed urinary frequency and dysuria. She received a domiciliary visit from her general practitioner 2 days ago and was prescribed trimethoprim for a possible urinary tract infection.

On arrival, she appears pale and clammy. Her peripheries are cold. Her observations reveal oxygen saturations of 94% on air, respiratory rate 28/min, heart rate 117/min, blood pressure 65/30 mmHg.

She has a past medical history of type 1 diabetes, uterine fibroids and hypothyroidism.

Initial investigations reveal:

White cell count	17.8 *109/l
Haemoglobin	97 g/l
Mean cell volume (MCV)	103.7 fL
Sodium	134 mmol/l
Potassium	4.9 mmol/l
Urea	7.0 mmol/l
Creatinine	120 µmol/l
Bilirubin	45 µmol/l
Alanine transaminase (ALT)	1051 U/l
Albumin	16 g/l
C-reactive protein (CRP)	71 mg/dL
Glucose	9.1 mmol/l

Urinalysis: ++protein, ++blood, +++leukocytes, ++nitrites, trace ketones

She is given intravenous fluids. Her blood pressure is 82/45 mmHg after a total of 3 litres of fluids. She is started on intravenous amoxicillin and gentamicin.

What is the next step in her management?

- ○ 1000mls intravenous colloid fluid ×
- ○ Intravenous hydrocortisone ×
- ○ Intravenous noradrenaline ×
- ○ Intravenous albumin ×
- ○ Intravenous meropenem ×

Submit answer

Reference ranges

Score: **69.7%**

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Albumin	16 g/l
C-reactive protein (CRP)	71 mg/dL
Glucose	9.1 mmol/l

Urinalysis: ++protein, ++blood, +++leukocytes, ++nitrites, trace ketones

She is given intravenous fluids. Her blood pressure is 82/45 mmHg after a total of 3 litres of fluids. She is started on intravenous amoxicillin and gentamicin.

What is the next step in her management?

1000mls intravenous colloid fluid	5%
[✓ Intravenous hydrocortisone]	74%
Intravenous noradrenaline	14%
Intravenous albumin	7%
Intravenous meropenem	1%

This patient has an autoimmune diathesis: she has type 1 diabetes and hypothyroidism. Her macrocytic anaemia also raises the possibility of undiagnosed pernicious anaemia. She is therefore at very high risk of primary adrenal insufficiency (Addison's Disease). IV hydrocortisone is a potentially life-saving treatment and should be given immediately. It is not contra-indicated in the presence of sepsis. Her biochemistry is not classical for Addison's, however, this does not exclude the diagnosis. Her abnormal LFTs are in keeping with a shocked liver. Intensive care review should be sought and she may well require inotropes but treatment of potential adrenal insufficiency is the immediate priority.

👍 👎 Discuss (13) Improve

Next question

Addisonian crisis

Causes
- sepsis or surgery causing an acute exacerbation of chronic insufficiency (e.g. Addison's, hypopituitarism)

- adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcaemia)
- steroid withdrawal (most common precipitant)
- inadequate 'sick day rule' management or intercurrent illness (commonly gastroenteritis) in patients with underlying Addison's disease

Signs and symptoms

- confusion, syncope, lethargy, convulsions
- hypotension, tachycardia, hypothermia
- hyponatraemia, hyperkalaemia, hypoglycaemia
- abdominal pain or cramps

Management

- hydrocortisone 100 mg IM or IV followed by 200 mg per 24 hours as a continuous infusion or 50 mg IV or IM every 6 hours (taper according to response as the patient becomes stable)
- 1 litre of normal saline infused over 30–60 minutes, then a further 4–6 litres over the first 24 hours if required; include dextrose if hypoglycaemic
- no fludrocortisone is required acutely as high-dose cortisol exerts a weak mineralocorticoid effect
- once stable, convert to oral replacement after approximately 24 hours, reducing to maintenance dosing over 3–4 days
- identify and treat the precipitating cause (e.g. infection)
- educate patients (and carers) about 'sick day rules' and encourage use of steroid alert cards or bracelets to prevent future crises

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 8 👎 2

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 6 👎 0

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 4 👎 0

Report broken media

Score: **70.6%**

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34 ✓

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Question 35 of 342

A 19-year-old woman is brought to the endocrinology clinic. She has had episodes of feeling weak and needing a sweet drink to improve. This has been occurring frequently since she was rejected from applications to medical school. She has had problems with anxiety previously, but the parents have become concerned as they have once used the patient's sister's glucose monitoring equipment, as she has type 1 diabetes, and found the patient to have capillary glucose as low as 2mmol/l. They have become concerned that the patient may have an insulinoma after reading about this on the internet, and therefore insisted on an endocrinology referral from the GP. Which investigation is best suited to exclude insulinoma?

- ○ CT abdomen ×
- ○ Capillary blood glucose monitoring over 48 hours ×
- ○ Post-prandial serum insulin and C-peptide ×
- ○ Supervised fasting with serum insulin and C-peptide ×
- ○ Synacthen test ×

Submit answer

Reference ranges

Score: **70.6%**

1 ✓

2	✓
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Question 35 of 342 ✓

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CT abdomen	1%
Capillary blood glucose monitoring over 48 hours	1%
Post-prandial serum insulin and C-peptide	9%
Supervised fasting with serum insulin and C-peptide	88%
Synacthen test	0%

Elevated C-peptide in fasting hypoglycaemia is suggestive of insulinoma

Important for me Less important

This patient has episodes of hypoglycaemia. There are several causes for hypoglycaemia, including insulinoma, liver failure, Addison's disease, alcohol and use of oral hypoglycaemic agents and insulin. Whilst insulinomas can cause episodes of hypoglycaemia, commonly associated with rapid weight gain and MEN-1, other explanations should also be considered. As this patient has a relative with type 1 diabetes she may have an opportunity to acquire insulin and use this herself. The most appropriate investigation to differentiate between the two is supervised fasting with monitoring of blood glucose, insulin and C-peptide can demonstrate or exclude insulinoma. If the patient were to have an insulinoma, then during fasting the blood glucose would become low, with elevated serum insulin and C-peptide, as C-peptide corresponds to endogenous rather than exogenous insulin. If the patient had hypoglycaemia during a fast with elevated insulin but low C-peptide the diagnosis would more likely be exogenous insulin use. A CT scan of the abdomen can be used if insulinoma is suspected following supervised fasting. Synacthen testing should be used to suspected Addison's disease.

👍 👎 Discuss (2) Improve

Next question

Hypoglycaemia ★

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
  - causes exaggerated insulin secretion
  - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia

- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later
- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic)

neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features
- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
  - Sweating
  - Shaking
  - Hunger
  - Anxiety
  - Nausea
- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
  - Weakness
  - Vision changes
  - Confusion
  - Dizziness
- Severe and uncommon features of hypoglycaemia include:
  - Convulsion
  - Coma

Investigation of hypoglycaemia
- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin	Insulinoma, Sulfonylurea use/abuse

Insulin Level	C-peptide Level	Interpretation	Potential Causes
		production	
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
  - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form
  - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
  - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
  - If the patient is alert, a quick-acting carbohydrate may be given (as above)
  - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
  - Alternatively, intravenous 20% glucose solution may be given through a large vein

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **71.4%**

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Question 36 of 342

A 52 year old lady presents complaining of polydipsia and polyuria. She has a background of hypertension, hypercholesterolaemia and bipolar affective disorder and a strong family history of diabetes - she is unsure which type.

Results show the following:

Na+	131mmol/l
urine osmolality	287mOsmol/kg (300 - 900mOsmol/kg)
plasma osmolality	287mOsmol/kg (285 - 295mOsmol/kg)

Which of the following is the most likely explanation for this lady's symptoms?

- ○ Psychogenic polydipsia ×
- ○ Syndrome of inappropriate anti-diuretic hormone (SIADH) ×
- ○ Diabetes insipidus ×
- ○ Diabetes mellitus type 1 ×
- ○ Hyponatraemia ×

Submit answer

Reference ranges

Score: **71.4%**

1	✓
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34 ✓

35 ✓

36 -

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Results show the following:

Na+	131mmol/l
urine osmolality	287mOsmol/kg (300 - 900mOsmol/kg)
plasma osmolality	287mOsmol/kg (285 - 295mOsmol/kg)

Which of the following is the most likely explanation for this lady's symptoms?

🟩	Psychogenic polydipsia	50%
	Syndrome of inappropriate anti-diuretic hormone (SIADH)	12%
	Diabetes insipidus	28%
🟥	Diabetes mellitus type 1	6%
	Hyponatraemia	4%

Although this lady is biochemically hyponatraemic, this is unlikely to be the cause of her symptoms.

With a past medical history of bipolar affective disorder, although not stated in the question, there is a good chance she may be on lithium which predisposes her to developing nephrogenic diabetes insipidus. However with this diagnosis, we would expect a much lower urine osmolality and a higher plasma osmolality. She would also have a normal to high serum sodium. The opposite would indicate a diagnosis of SIADH (serum hypo-osmolality and high urine osmolality).

The osmolality results here reflect a diagnosis of psychogenic polydipsia with a low urine osmolality and a low end of normal plasma osmolality.

👍 👎 Discuss (11) Improve

Next question

Hyponatraemia

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)
- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic
- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss
- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

+ Q 123

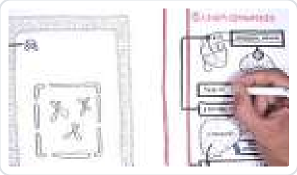
Next question

Textbooks

High-yield textbook

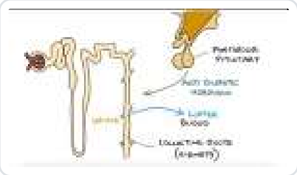
Extended textbook

Media



Hyponatraemia - classification, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 5 👎 0



Understanding Hyponatraemia

Zero To Finals - YouTube

👍 17 👎 1

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **69.4%**

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Question 37 of 342

A 47-year-old woman with a history of paroxysmal atrial fibrillation undergoes a thyroidectomy for Graves' disease. Initially, her symptoms settle. However, on postoperative scans, residual thyroid tissue is noted and after several months, her symptoms recur, including palpitations from atrial fibrillation. She is referred for radioiodine therapy.

Three days before the radioiodine administration is due, she is admitted with acute-onset abdominal pain and, given the history of atrial fibrillation, ischaemic colitis is suspected. An urgent CT scan of the abdomen is performed with contrast, which rules out any acute pathology and so she is discharged.

With regards to the radioiodine therapy, when is the earliest that this can take place?

- ○ 3 days ✕
- ○ 2 weeks ✕
- ○ 8 weeks ✕
- ○ 16 weeks ✕
- ○ 26 weeks ✕

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **69.4%**

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With regards to the radioiodine therapy, when is the earliest that this can take place?

	3 days	12%
🟥	2 weeks	42%
🟩	8 weeks	40%
	16 weeks	5%
	26 weeks	1%

Radioiodine therapy should be avoided until 8 weeks following CT contrast administration

Important for me Less important

Iodine in intravenous contrast interferes with the administration of radioiodine. As the levels of iodine in contrast are several hundred-fold higher than the recommended daily allowance, this may be retained by the body for months. As such, it can interfere with therapeutic uptake of radioiodine by the thyroid gland. Various studies have demonstrated a 6-8 week time period is the minimum to wait before administering radioiodine, following CT contrast. **8 weeks** is therefore the correct answer.

Going ahead with the procedure as planned, **3 days** after this CT scan with contrast is therefore too soon. The contrast would interfere with the radioiodine.

Similarly, **2 weeks** is still too soon to go ahead with the administration.

16 weeks is an unnecessarily long amount of time to wait - 8 weeks is sufficient according to current studies.

Similarly, **26 weeks** is longer than needed - the question asks what the earliest time of administration should be.

👍 👎 💬 Discuss (6) Improve

Next question

Radioiodine therapy ★

Nearly all the iodine ingested is taken up by the thyroid gland. It is an essential element for the formation of T3 and T4. Radioiodine therapy involves using radioactive iodine (I-131 isotope). The radiation can destroy the thyroid gland and any other thyroid cells (including cancer cells) that take up iodine, with little effect on the rest of the body.

Indications
- Differentiated thyroid cancer
- Toxic multinodular goitre
- Graves disease refractory to medical management
- Radiation exposure: Potassium iodine has been used to help individuals exposed to radiation by reducing the harmful accumulation of radioactive substances in the thyroid

Method

- TSH is required for iodine uptake by thyroid cells. In order to effectively treat with radioactive iodine, TSH levels have to be increased to stimulate any thyroid cells into taking up the radioactive iodine. This is done by either making patients hypothyroid after stopping thyroid hormone or by treating with recombinant human TSH.
- A capsule of radioactive iodine is ingested. A clear perspex tube is used to transport the capsule into the patient's mouth to avoid skin contact.
- Once the capsule has been ingested the patient can leave the isotopes unit.

Advice post procedure

- After radioactive iodine, patients should be advised to keep babies, children under five, pregnant women and pets at arm's length for two to three weeks
- It is important not to become pregnant for at least six months after radioactive iodine treatment
- Male patients are advised not to cause a pregnancy for six months after radioactive iodine

Contraindications

- Pregnancy
- Breastfeeding
- Active thyroid eye disease (unless providing steroid cover)

Complications

- Thyroid pain
- Hypothyroidism
- Flare of Graves' eye disease

Follow up

- Monitoring of patients thyroid function (hypothyroidism is the commonest complication of radioiodine therapy)
- For thyroid cancers, monitoring of thyroglobulin. If thyroglobulin concentrations are initially elevated in a person diagnosed with

thyroid cancer, then it is likely that thyroglobulin can be used as a tumour marker.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **67.6%**

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← Question 38 of 342 ⚐ →

A 37-year-old female presents with 4 days of generally unwell and a recent dysuria. Her urine is foul smelling and dark. She is a known type 1 diabetic with a long-standing subcutaneous insulin regime. Her pH on admission was 7.24, bicarbonate 8 mmol/l and blood glucose 32 mmol/l. Urinary dip leucocytes 2+, nitrites2+ and 4+ ketones. She was started on treatment for diabetic ketoacidosis with intravenous fluids and fixed rate insulin. She also has intravenous antibiotics for a urinary source of sepsis. You are asked to review her blood sugars at 4 hours after treatment was initiated. What should be the aim in managing hyperglycaemia in a diabetic ketoacidosis patient?

○ Reduce blood glucose to under 14 mmol/l as quickly as possible ✕

○ Reduce blood glucose by 3mmol/l per hour ✕

○ Reduce blood glucose by 6 mmol/l per hour ✕

○ Aim blood glucose above 18 mmol/l ✕

○ Blood glucose does not require monitoring if insulin infusion is running ✕

Submit answer

Reference ranges ⌄

Score: **67.6%**

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Question 38 of 342 ✓

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Reduce blood glucose to under 14 mmol/l as quickly as possible	14%
Reduce blood glucose by 3mmol/l per hour	61%
Reduce blood glucose by 6 mmol/l per hour	18%
Aim blood glucose above 18 mmol/l	3%
Blood glucose does not require monitoring if insulin infusion is running	3%

The most recent guidelines by the Joint British Diabetes Societies Inpatient Care Group in September 2012 recommends a reduction in blood glucose of 3 mmol/l per hour until BM reaches 14 mmol/l, at which point 5% dextrose should be considered as the intravenous fluid of choice. Rapid glucose lowering should be avoided: the rapid flux in osmolality can result in significant cerebral oedema and resultant cerebral damage.

Discuss (3) Improve

Next question

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l

American Diabetes Association (2009)	Joint British Diabetes Societies (2013)
- anion gap > 10 - ketonaemia	- ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour

Fluid	Volume
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

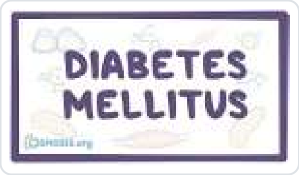
Report broken link

Media

Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **68.4%**

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Question 39 of 342

A 28-year-old lady is diagnosed with gestational diabetes in her first pregnancy. Her fasting blood glucose is 5.9mmol/l and blood glucose after oral glucose tolerance test (OGTT) is 8.2mmol/l. Blood glucose control during pregnancy is achieved with diet, exercise and metformin. She gives birth to a healthy child at 39 weeks. A fasting blood glucose at day 1 post-partum is 5.2mmol/l.

Which of the following statements is correct with respect to follow-up monitoring for diabetes?

- ○ OGTT 6-13 weeks postpartum ×
- ○ Fasting blood glucose test 6-13 weeks postpartum ×
- ○ No routine follow up unless further pregnancy ×
- ○ HbA1c 6-13 weeks postpartum ×
- ○ Annual fasting blood glucose checks only ×

Submit answer

Reference ranges

Score: **68.4%**

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Which of the following statements is correct with respect to follow-up monitoring for diabetes?

	OGTT 6-13 weeks postpartum	34%
🟩	Fasting blood glucose test 6-13 weeks postpartum	35%
	No routine follow up unless further pregnancy	14%
🟥	HbA1c 6-13 weeks postpartum	12%
	Annual fasting blood glucose checks only	5%

Women with gestational diabetes whose glucose returns to normal after birth need a postnatal glucose check 6-13 weeks postpartum to stratify their risk of developing diabetes in the future. NICE recommends that this is a fasting blood glucose. Further follow up will depend on the result of this postnatal check. Even if postnatal glucose is less than 6mmol/l, annual fasting glucose checks are still recommended thereafter.

Discuss (6) | Improve

Next question

Gestational diabetes ★

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:

- 87.5% have gestational diabetes
- 7.5% have type 1 diabetes
- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes

- BMI of > 30 kg/m^2
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes
- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)
- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:

- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose
- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
    - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
    - if glucose targets are still not met insulin should be added to diet/exercise/metformin
    - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios, insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets with metformin but decline insulin treatment

Management of pre-existing diabetes

- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	Target
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link

Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

Report broken media

Score: **66.7%**

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You are asked to review a 55-year-old male surgical for the fourth time in seven days with persistent hyperkalaemia on his blood tests. He has been admitted for 5 weeks under the surgeons following AP resection of sigmoid carcinoma complicated by a superficial wound infection requiring a vacuum dressing. In all of the previous three medical reviews, the patient presented with a serum potassium of greater than 6.5 mmol/l and was treated with insulin-dextrose and calcium gluconate.

His past medical history includes type 2 diabetes mellitus, non-alcoholic steatohepatitis and neuromyelitis optica diagnosed 6 years ago and stable on the last review 2 months ago. His regular medications include gliclazide 80mg BD, Lantus (insulin glargine) 15 units OD, prednisolone 15 mg OD and baclofen 10mg QDS. During this review, he is alert and comfortable, blood pressure measures 135/82 mmHg, heart rate 90/min and sinus.

His blood tests are as follows:

Hb	121 g/l
Platelets	334 * 10^9/l
WBC	8.2 * 10^9/l

Na^+	136 mmol/l
K^+	6.9 mmol/l
Urea	7.5 mmol/l
Creatinine	110 µmol/l
CRP	4 mg/l
Renin	Raised
Aldosterone	Decreased

Blood gases show the following:

pH	7.24
PaO2 (air)	15.8 kPa
PaCO2	2.2 kPa
Bicarbonate	24 mmol/l

Urinary pH = 6.2

A repeat CT abdomen and pelvis demonstrates appropriate wounding healing with no local collections at the resection site. No other abdominal pathology is noted.

What is the most likely diagnosis?

- ○ Type 1 renal tubular acidosis ✕
- ○ Type 2 renal tubular acidosis ✕
- ○ Type 4 renal tubular acidosis ✕
- ○ Waterhouse-Friderichsen syndrome ✕
- ○ Addisonian crisis ✕

Submit answer

Reference ranges

Score: **66.7%**

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Aldosterone	Decreased

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PaCO2	2.2 kPa
Bicarbonate	24 mmol/l

Urinary pH = 6.2

A repeat CT abdomen and pelvis demonstrates appropriate wounding healing with no local collections at the resection site. No other abdominal pathology is noted.

What is the most likely diagnosis?

Type 1 renal tubular acidosis	3%
Type 2 renal tubular acidosis	2%
[✓ Type 4 renal tubular acidosis]	82%
Waterhouse-Friderichsen syndrome	6%
Addisonian crisis	7%

Refractory hyperkalaemia in a patient with a prolonged illness should raise suspicions for adrenal insufficiency. Note that mineralocorticoid deficiency can occur with hyperkalaemia alone without hyponatraemia. In this case, the serum demonstrates a metabolic acidosis with normal bicarbonate and urinary pH greater than 5.5, ruling out type 1 and 2 renal tubular acidosis (RTA). Waterhouse-Friedrichsen syndrome is caused by adrenal haemorrhage, classically secondary to tuberculosis or meningococcal infection, which if present, should be visualised on CT imaging. He does not demonstrate circulatory collapse, abdominal pain or nausea suggestive of Addisonian crisis. Type 4 RTA, causing a failure of the sodium-potassium antiporter is thus the most appropriate diagnosis, in the context of a patient with chronic steroid use and hence predisposition for adrenal insufficiency during acute severe illness.

Discuss (14) | Improve

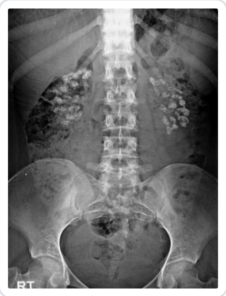
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

RENAL TUBULAR ACIDOSIS
Osmosis.org

Renal tubular acidosis

Osmosis - YouTube 8 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **67.5%**

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Question 41 of 342

A 54-year-old woman presents to the emergency department with sweating and palpitations. She has a past medical history of Graves disease. She has completed a course of carbimazole approximately six months ago. Her other past medical history includes type 2 diabetes, hypertension, ischaemic heart disease and morbid obesity. She is on metformin, aspirin, amlodipine and atorvastatin.

Her observations are as follows:

- Heart rate 111 beats per minute
- Blood pressure 124/72 mmHg
- Respiratory rate 21/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

On examination, she is diaphoretic and anxious. A tremor is noted. Ophthalmic examination is normal. There is a very small goitre. Her body mass index is 53 kg/kg/m².

Blood tests:

TSH	0.0 mIU/L	(0.2-5.5)
Free T4	42 pmol/L	(10 - 24.5)

Given the likely diagnosis, what is the optimal treatment approach to address the underlying cause?

○ Propranolol ×

○ Propylthiouracil ×

○ Radioiodine therapy ×

○ Repeat carbimazole treatment ✕

○ Surgery ✕

Submit answer

Reference ranges ⌵

Score: **67.5%**

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Blood tests:

TSH	0.0 mIU/L	(0.2-5.5)
Free T4	42 pmol/L	(10 - 24.5)

Given the likely diagnosis, what is the optimal treatment approach to address the underlying cause?

Propranolol	18%
Propylthiouracil	15%
Radioiodine therapy	52%

Repeat carbimazole treatment	7%
Surgery	8%

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Important for me Less important

Radioiodine therapy is the correct answer. This patient has a relapse of Graves' disease. Guidelines recommend that in those patients who have a relapse of disease despite a course of antithyroid drug treatment, definitive management should be instituted in the form of radioiodine treatment or surgery. This patient's many medical problems including ischaemic heart disease and morbid obesity make her a significant anaesthetic risk and therefore radioiodine therapy would be preferable.

Surgery is incorrect. This is an option for the treatment of relapsed Graves' disease. However, she would be a high-risk surgical candidate and therefore radioiodine therapy would be preferable.

Repeat carbimazole treatment is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves' disease.

Propylthiouracil is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves' disease.

Propranolol is incorrect. This would control some of the symptoms of hyperthyroidism without treating the underlying cause.

👍 👎 💬 Discuss (9) Improve

Next question

Graves' disease: management

Despite many trials, there is no clear guidance on the optimal management of Graves' disease. Treatment options include anti-thyroid drugs (ATDs, for example carbimazole), radioiodine treatment and surgery.

Anti-thyroid drugs have emerged as the most popular first-line therapy for Graves' disease in recent years. Factors that particularly support their use include anti-thyroid drugs significant symptoms of thyrotoxicosis, or patients with a significant risk of hyperthyroid complications (e.g. elderly patients, cardiovascular disease).

Initial treatment to control symptoms
- propranolol is used to help block the adrenergic effects

NICE recommends that patients with Graves' disease are referred to secondary care for ongoing treatment.
- NICE suggest carbimazole should be considered in primary care if patients symptoms are not controlled with propanolol

ATD therapy
- carbimazole is started at 40mg and reduced gradually to maintain euthyroidism
- typically continued for 12-18 months
- the major complication of carbimazole therapy is agranulocytosis
- an alternative regime is termed 'block-and-replace'
  - carbimazole is started at 40mg
  - thyroxine is added when the patient is euthyroid
  - treatment typically lasts for 6-9 months
  - patients following an ATD titration regime have been shown to suffer fewer side-effects than those on a block-and-replace regime

Radioiodine treatment
- often used in patients who relapse following ATD therapy or are resistant to primary ATD treatment

- contraindications include pregnancy (should be avoided for 4-6 months following treatment) and age < 16 years. Thyroid eye disease is a relative contraindication, as it may worsen the condition
- the proportion of patients who become hypothyroid depends on the dose given, but as a rule the majority of patient will require thyroxine supplementation after 5 years

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

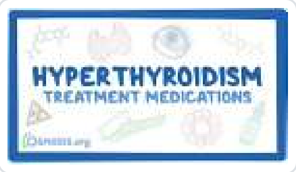
Links

Clinical Knowledge Summaries 11 4

Graves' disease: management

Suggest link Report broken link

Media



Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 0

ENDOCRINOLOGY
GRAVES DISEASE

Graves' disease

Armando Hasudungan - YouTube 👍 2 👎 2

Report broken media

Score: **65.9%**

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Question 42 of 342

A 28-year-old woman is referred by her GP with refractory hypertension. Despite combination therapy with ramipril, amlodipine, bendroflumethiazide and atenolol, her blood pressure in clinic today is 181/105 mmHg. Some of her bloods are shown below. On direct questioning she also admits passing urine more than 10 times per day. What is the most likely diagnosis?

Na^+	145 mmol/l
K^+	3.0 mmol/l
Urea	6.0 mmol/l
Creatinine	71 µmol/l

What is the most likely diagnosis?

- ○ Phaeochromocytoma ✕
- ○ Coarctation of the aorta ✕
- ○ Renal artery stenosis ✕
- ○ 21-hydroxylase deficiency ✕
- ○ Conn's syndrome ✕

Submit answer

Reference ranges

Score: **65.9%**

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Na^+	145 mmol/l
K^+	3.0 mmol/l
Urea	6.0 mmol/l
Creatinine	71 µmol/l

What is the most likely diagnosis?

	Phaeochromocytoma	9%
	Coarctation of the aorta	0%
🟥	Renal artery stenosis	7%
	21-hydroxylase deficiency	6%
🟩	Conn's syndrome	77%

All of the answers above are causes of secondary hypertension, except for 21-hydroxylase deficiency which accounts for over 90% of congenital adrenal hyperplasia.

The high sodium and low potassium in the bloods reflects the increased levels of aldosterone produced in Conn's syndrome. Aldosterone stimulates the Na+/K+-ATPase membrane transporter in the distal convoluted tubule resulting in increased sodium reabsorption and potassium excretion. Polyuria and polydipsia can result from the kidneys inability to concentrate urine.

The diagnosis is usually confirmed using plasma aldosterone:renin ratio

👍 👎 Discuss (10) Improve

Next question

Primary hyperaldosteronism

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes
- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

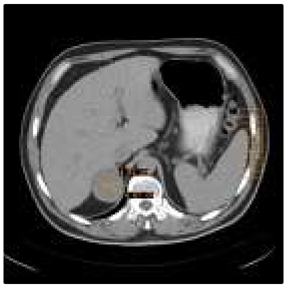
Features
- **hypertension**
  - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
  - e.g. muscle weakness
  - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
    - hypertension with hypokalemia
    - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
    - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
    - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

Suggest link

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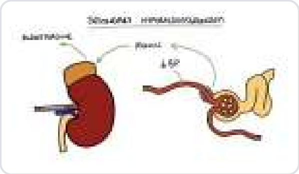
Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

Score: **64.3%**

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← Question 43 of 342 →

A 65-year-old man presents to his general practitioner with polyuria and polydipsia. He has a past medical history of surgically treated bladder cancer, hypertension and chronic kidney disease. His medications include amlodipine, furosemide, atenolol and ramipril.

The examination is unremarkable.

Blood tests:

Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	28.2 mmol/L	(2.0 - 7.0)
Creatinine	312 µmol/L	(55 - 120)
eGFR	21 ml/minute	(>90)
HbA1c	52 mmol/mol	(<48)

What is the most appropriate medication to commence?

- ○ Empagliflozin ✕
- ○ Insulin ✕
- ○ Metformin ✕
- ○ Pioglitazone ✕
- ○ Sitagliptin ✕

Submit answer

Reference ranges

Score: **64.3%**

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What is the most appropriate medication to commence?

🟥	Empagliflozin	37%
	Insulin	18%
	Metformin	9%
	Pioglitazone	2%
🟩	Sitagliptin	33%

T2DM initial therapy: if metformin is contraindicated (and no risk of CVD, established CVD or chronic heart failure) → choice of DPP-4 inhibitor or Pioglitazone or Sulfonylurea or even SGLT-2 (if NICE criteria met)

Important for me Less important

Sitagliptin is correct. The patient has symptoms and biochemical evidence of type 2 diabetes with an hba1c level > 48 mmol/mol. Typically, patients would be treated with oral anti-diabetic medications in the first instance. Metformin is the usual choice but it is contraindicated in severe renal impairment. There are a number of other medications that can be chosen depending on the patient's profile. Many of them are contraindicated in severe renal impairment. Sitagliptin is the medication that is most appropriate as it can be started 1st line for T2DM when metformin is contraindicated. It can be started at a reduced dose when the eGFR is < 30ml/minute.

Metformin is incorrect. This is contraindicated in severe renal impairment.

Empagliflozin is incorrect. This is typically not initiated in type 2 diabetes if the eGFR is < 45ml/minute.

Insulin is incorrect. This is not typically used 1st line in with the management of type 2 diabetes as oral medications are typically trialled in the first instance.

Pioglitazone is incorrect. This is contraindicated in those patients with a history of bladder cancer.

👍 👎 Discuss (7) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

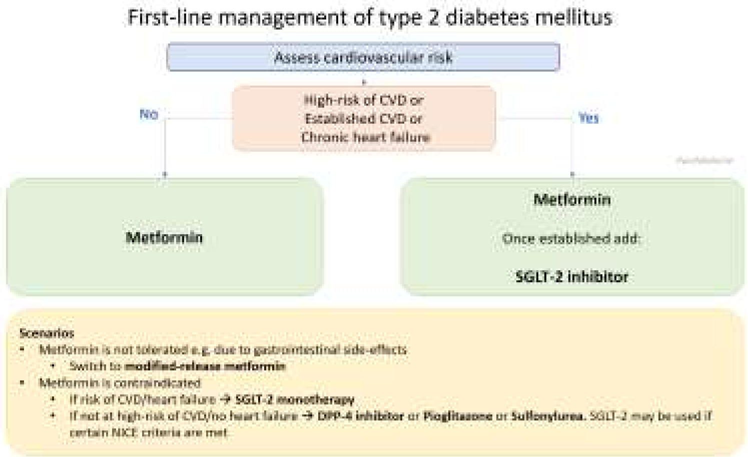
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios

- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

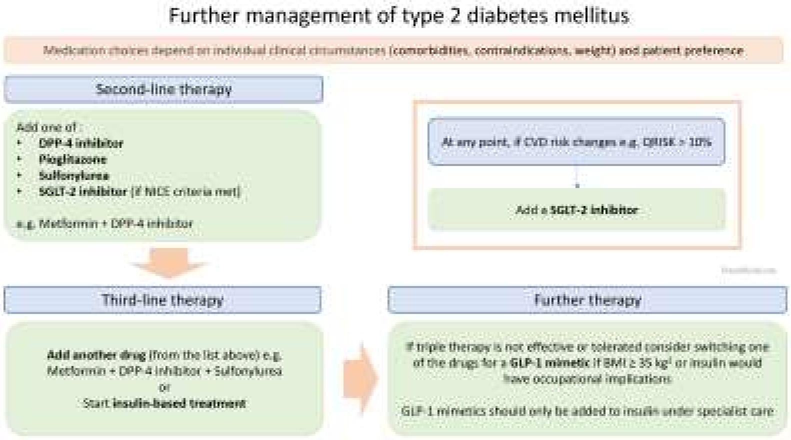
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

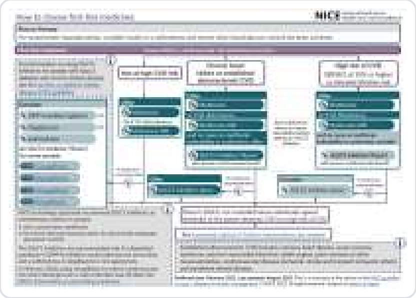
Starting insulin

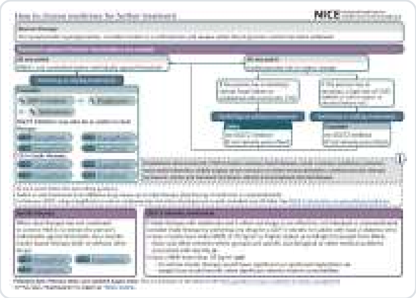
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea

Type 2 diabetes in adults: choosing medicines





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

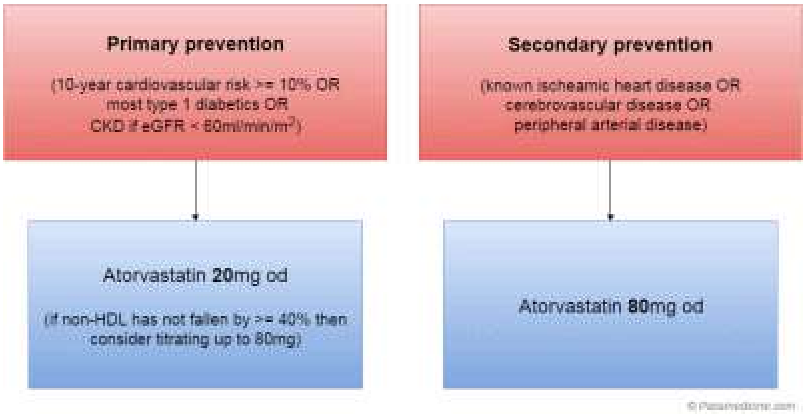
	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischaemic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

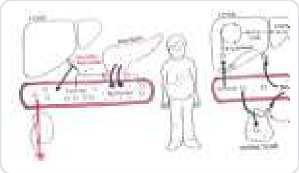
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

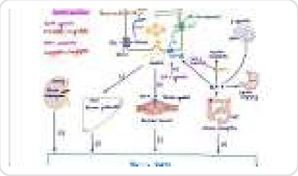
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **62.8%**

1 ✓
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Dr. Jamil Alrajhi

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← Question 44 of 342 →

A 28-year-old woman is referred to the hospital with dysphagia. She complains of fatigue and that she often struggles to swallow certain foods such as bread. Drinking has not been a problem.

In addition, her eyes often feel gritty and uncomfortable. She has mild myalgia which interferes with her job at the local bakery.

What other finding would be consistent with the most likely diagnosis?

- ○ C-ANCA positive ✕
- ○ P-ANCA positive ✕
- ○ Type 1 renal tubular acidosis ✕
- ○ Type 2 renal tubular acidosis ✕
- ○ Type 4 renal tubular acidosis ✕

Submit answer

Reference ranges

Score: **62.8%**

1 ✓
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4 ✓

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Question 44 of 342 ✓

A 28-year-old woman is referred to the hospital with dysphagia. She complains of fatigue and that she often struggles to swallow certain foods such as bread. Drinking has not been a problem.

In addition, her eyes often feel gritty and uncomfortable. She has mild myalgia which interferes with her job at the local bakery.

What other finding would be consistent with the most likely diagnosis?

C-ANCA positive	4%
P-ANCA positive	5%
Type 1 renal tubular acidosis	74%
Type 2 renal tubular acidosis	14%
Type 4 renal tubular acidosis	2%

Type 1 (**distal**) renal tubular acidosis may be caused by Sjogren's syndrome

Important for me Less important

This patient has features consistent with Sjogren's syndrome. The xerostomia (dry mouth) can lead to difficulty swallowing, particularly foods needing more saliva. The other characteristic feature is xerophthalmia (dry eyes) which can make the eyes feel gritty and uncomfortable. Sjogren's syndrome is associated with **type 1 renal tubular acidosis**.

Type 1 (distal) renal tubular acidosis occurs due to an inability to excrete H+ in the distal tubule. The resulting acidosis is usually accompanied by hypokalaemia. Complications include nephrocalcinosis and renal stones. Causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, and analgesic nephropathy.

A sometimes helpful reminder is that type 1 renal tubular acidosis is largely associated with structural/rheumatological problems, whereas, **type 2 renal tubular acidosis** is associated with metabolic disorders such as Fanconi syndrome, Wilson's disease, and cystinosis.

Type 4 renal tubular acidosis is associated with hypoaldosteronism and diabetes, rather than Sjogren's. Type 4 is associated with hyperkalaemia, whilst types 1 and 2 are associated with hypokalaemia.

Sjogren's is typically associated with elevated levels of anti-Ro and anti-La. Neither **cANCA** nor **pANCA** are typically associated. ANCA antibodies are commonly associated with vasculitides. cANCA (cytoplasmic-ANCA) is associated with granulomatosis with polyangiitis. pANCA is found in microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis.

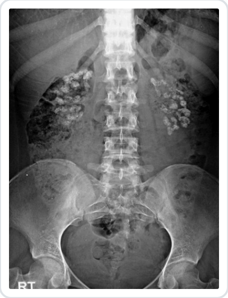
👍 👎 Discuss (2) Improve

Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

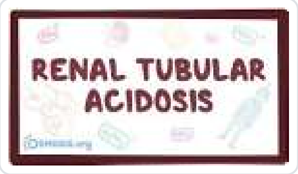
Next question

Textbooks

High-yield textbook

Extended textbook

Media


RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **63.6%**

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43 ✗

44 ✓

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Question 45 of 342

A 62-year-old man presents to the emergency department with vomiting and abdominal pain. He has been becoming worse over four days since eating a barbecued piece of chicken he suspects was undercooked. Since then he has had diarrhoea and vomiting and has recently been noticing abdominal pain. His wife brought him to the emergency department as she felt he was starting to become drowsy. He has a past medical history of type 2 diabetes mellitus, osteoarthritis, steatohepatitis and stable angina. He normally takes metformin, dapagliflozin, aspirin, omeprazole, paracetamol and bisoprolol. Investigations reveal that he has a serum glucose of 12.1 mmol/L, a pH of 6.9 and urine testing reveal +++ of ketones. He is diagnosed with euglycaemic diabetic ketoacidosis. What factor has most likely contributed to the development of diabetic ketoacidosis?

○ Drug history of dapagliflozin ×

○ Drug history of metformin ×

○ Drug history of bisoprolol ×

○ Medical history of steatohepatitis ×

○ Medical history of angina ×

Submit answer

Reference ranges

Score: **63.6%**

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Question 45 of 342 ✓

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Drug history of dapagliflozin	88%
Drug history of metformin	6%
Drug history of bisoprolol	1%
Medical history of steatohepatitis	3%
Medical history of angina	2%

SGLT2 inhibitors can cause normoglycaemic ketosis in type 2 diabetes

Important for me Less important

The most likely explanation is the drug history of dapagliflozin. Dapagliflozin is a SGLT2 inhibitor which reduces glucose reabsorption in the urine and increases urinary glucose excretion. This patient has developed one of the side effects of dapagliflozin; DKA. DKA secondary to SGLT2 dapagliflozin is more likely to be euglycaemic than DKA in general. Metformin is associated with metabolic acidosis, especially in the context of reduced renal function or alcohol, and bisoprolol can cause hypoglycaemia. Steatohepatitis can eventually result in hepatic failure causing impaired gluconeogenesis, but this is more likely to result in hypoglycaemia rather than DKA. Angina is unlikely to be relevant.

👍 👎 Discuss (5) Improve

Next question

SGLT-2 inhibitors ★

SGLT-2 inhibitors reversibly inhibit sodium-glucose co-transporter 2 (SGLT-2) in the renal proximal convoluted tubule to reduce glucose reabsorption and increase urinary glucose excretion.

Examples include canagliflozin, dapagliflozin and empagliflozin.

Important adverse effects include
- urinary and genital infection (secondary to glycosuria). Fournier's gangrene has also been reported
- normoglycaemic ketoacidosis
- increased risk of lower-limb amputation: feet should be closely monitored

Patients taking SGLT-2 drugs often lose weight, which can be beneficial in type 2 diabetes mellitus.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

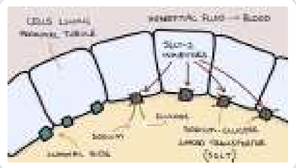
Links

BNF 👍 14 👎 15

Dapagliflozin

Suggest link Report broken link

Media


CELLS LINING PROXIMAL TUBULE
INTERSTITIAL FLUID → BLOOD
SGLT-2 INHIBITORS
GLUCOSE
SODIUM
LUMINAL SIDE
SODIUM-GLUCOSE LINKED TRANSPORTER (SGLT)

How does Dapagliflozin work? Understanding SGLT2 inhibitors.

Zero To Finals - YouTube 👍 6 👎 0

Report broken media

Score: **64.4%**

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Question 46 of 342

A 27-year-old woman who is 11 weeks pregnant comes for review. This is her second pregnancy. During her first pregnancy she was diagnosed with gestational diabetes which resolved following the birth of her son. What is the most appropriate management at this stage?

- ○ Perform an oral glucose tolerance test ✕
- ○ Advise on a diabetic diet and start metformin at 20 weeks ✕
- ○ Arrange a fasting glucose ✕
- ○ Arrange a HbA1c test ✕
- ○ Advise on a diabetic diet and start insulin at 20 weeks ✕

Submit answer

Reference ranges

Score: **64.4%**

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Question 46 of 342 ✓

A 27-year-old woman who is 11 weeks pregnant comes for review. This is her second pregnancy. During her first pregnancy she was diagnosed with gestational diabetes which resolved following the birth of her son. What is the most appropriate management at this stage?

Perform an oral glucose tolerance test	80%
Advise on a diabetic diet and start metformin at 20 weeks	3%
Arrange a fasting glucose	11%
Arrange a HbA1c test	3%
Advise on a diabetic diet and start insulin at 20 weeks	3%

The oral glucose tolerance test remains the investigation of choice for gestational diabetes

Important for me Less important

Discuss (9) Improve

Next question

Gestational diabetes

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:

- 87.5% have gestational diabetes

- 7.5% have type 1 diabetes
- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes

- BMI of > 30 kg/m^2
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes
- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)
- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:
- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose

- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
  - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
  - if glucose targets are still not met insulin should be added to diet/exercise/metformin
  - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios, insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets with metformin but decline insulin treatment

Management of pre-existing diabetes

- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	**Target**
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

Report broken media

Score: **65.2%**

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Question 47 of 342

A 30-year-old male with background of type one diabetes mellitus presents with abdominal pain and shortness of breath. Investigations confirm he has diabetic ketoacidosis. Which one of the following investigations would suggest a discussion for possible intensive care admission?

○ Lactate 3 mmol/L ×

○ Bicarbonate level 19 mmol/L ×

○ pH 7.27 ×

○ White cell count 30 x 10^9/L ×

○ Potassium 3.4 mmol/L ×

Submit answer

Reference ranges

Score: **65.2%**

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← Question 47 of 342 ✗ →

A 30-year-old male with background of type one diabetes mellitus presents with abdominal pain and shortness of breath. Investigations confirm he has diabetic ketoacidosis. Which one of the following investigations would suggest a discussion for possible intensive care admission?

	Lactate 3 mmol/L	14%
	Bicarbonate level 19 mmol/L	5%
🟥	pH 7.27	41%
	White cell count 30 x 10^9/L	12%
🟩	Potassium 3.4 mmol/L	28%

Parameters indicate severe diabetic ketoacidosis:

- pH < 7
- Blood ketone > 6 mmol/L
- Bicarbonate < 5 mmol/L
- Anion gap >16 mmol/l
- Potassium < 3.5 mmol/L on admission
- Tachycardia or bradycardia
- Systolic blood pressure <90 mmHg
- Oxygen saturation <92% on air
- GCS < 12

Discuss (8) | Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours

Fluid	Volume
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist

- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

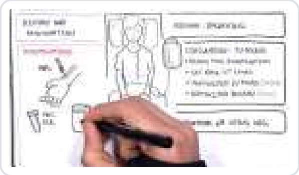
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

Report broken link

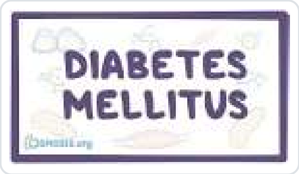
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **63.8%**

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A 39-year-old woman has undergone a total thyroidectomy for a papillary thyroid tumour which was 3.2cm in size without lymph node or metastatic involvement and negative margins. She is reviewed in the endocrinology clinic following her treatment and would like to know how best she should be monitored for recurrence of malignancy. What is the most appropriate monitoring?

- ○ Annual CT neck ×
- ○ Annual US neck ×
- ○ Annual MRI neck ×
- ○ Annual thyroglobulin ×
- ○ Annual TSH and free T4 ×

Submit answer

Reference ranges

Score: **63.8%**

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Question 48 of 342 ✗

A 39-year-old woman has undergone a total thyroidectomy for a papillary thyroid tumour which was 3.2cm in size without lymph node or metastatic involvement and negative margins. She is reviewed in the endocrinology clinic following her treatment and would like to know how best she should be monitored for recurrence of malignancy. What is the most appropriate monitoring?

	Annual CT neck	2%
	Annual US neck	10%
	Annual MRI neck	2%
🟩	Annual thyroglobulin	79%
🟥	Annual TSH and free T4	8%

Following surgery and radioiodine therapy, patients with papillary thyroid cancer should be monitored with thyroglobulin annually

Important for me Less important

This patient has a very good prognosis as she has had papillary thyroid cancer which is less than 4cm in size without lymph node or other organ involvement. The most appropriate way to monitor for cancer recurrence is annual thyroglobulin. Imaging is unlikely to detect a recurrence early and is therefore inappropriate. TSH and free T4 levels should be measured frequently as this patient will need thyroxine replacement, but this is not to detect recurrence of malignancy.

Discuss (3) Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	**Notes**
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated

Type	**Notes**
	• Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

Suggest link

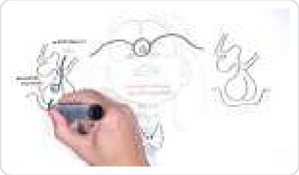
Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0



Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **62.5%**

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Question 49 of 342

A 78-year-old man with metastatic lung carcinoma presents with increasing lethargy and a number of falls. He describes feeling very unsteady on standing from his bed. His appetite has been poor and he has vomited two times each day for the past three days. There is no diarrhoea or abdominal pain and he denies any shortness of breath. He takes regular paracetamol for pain and omeprazole for dyspepsia.

On examination, he is a tanned gentleman with cachexia. He appears pale with dry mucosa and his abdomen is soft and non-tender. There is reduced air entry at the right base with bronchial breathing overlying. His heart sounds are normal and his capillary refill time is prolonged to 4 seconds. Bloods pressure is 85/65mmHg, heart rate 86/min, respiratory rate 23/min

Hb	102 g/l	Na$^+$	129 mmol/l
Platelets	189 * 10^9/l	K$^+$	5.0 mmol/l
WBC	5.6* 10^9/l	Urea	7.2 mmol/l
Neuts	4.2 * 10^9/l	Creatinine	87 µmol/l
Lymphs	0.7 * 10^9/l	CRP	32 mg/l
Eosin	0.1 * 10^9/l		

CT-chest-abdomen-pelvis	There is a 4cm mass in the right lower lobe with extension to adjacent pleura. Bilateral pleural effusions are present. There is mediastinal lymphadenopathy and enlargement of para-aortic nodes with masses seen in both adrenals. Findings are in keeping with a primary lung malignancy with metastatic spread

What is the likeliest explanation of this presentation?

- ○ SIADH ×
- ○ Terminal decline ×

○ Addisonian crisis ×

○ Proton pump inhibitor side effects ×

○ Pneumonia ×

Submit answer

Reference ranges

Score: **62.5%**

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Question 49 of 342 ✗

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What is the likeliest explanation of this presentation?

SIADH 27%

Terminal decline 3%

Addisonian crisis	69%
Proton pump inhibitor side effects	1%
Pneumonia	0%

Metastatic malignancy can cause Addison's disease

Important for me Less important

This gentleman presents in a shock like state with poor peripheral perfusion. This could be caused by pneumonia but there are no signs of sepsis and no consolidation on CT. SIADH and PPIs can cause hyponatraemia but not cardiovascular collapse. Terminal decline is the diagnosis of exclusion. This gentleman does have metastatic deposits in both adrenal glands which can impair function and cause Addison's. Furthermore, he has hyponatraemia, hyperkalaemia and hyperpigmentation all which are features seen in Addison's.

👍 👎 Discuss (4) Improve

Next question

Addison's disease ★

Autoimmune destruction of the adrenal glands is the most common cause of primary hypoadrenalism in the UK, accounting for 80% of cases. This is termed Addison's disease and results in reduced cortisol and aldosterone being produced.

Features

- lethargy, weakness, anorexia, nausea & vomiting, weight loss, 'salt-craving'
- hyperpigmentation (especially palmar creases)
    - ACTH is derived from a larger precursor molecule called proopiomelanocortin (POMC). When POMC is cleaved to produce ACTH, other melanocyte-stimulating hormones (MSH)

    are also produced. These MSHs have the effect of stimulating melanocytes in the skin to produce more melanin, the pigment responsible for skin colour
    - primary Addison's is associated with hyperpigmentation whereas secondary adrenal insufficiency is not
- vitiligo
- loss of pubic hair in women
- hypotension
- hypoglycaemia
- hyponatraemia and hyperkalaemia may be seen
- crisis: collapse, shock, pyrexia

Other causes of hypoadrenalism

Primary causes
- tuberculosis
- metastases (e.g. bronchial carcinoma)
- meningococcal septicaemia (Waterhouse-Friderichsen syndrome)
- HIV
- antiphospholipid syndrome

Secondary causes
- pituitary disorders (e.g. tumours, irradiation, infiltration)

Exogenous glucocorticoid therapy

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 10 👎 16

2017 Adrenal insufficiency â€“ recognition and management

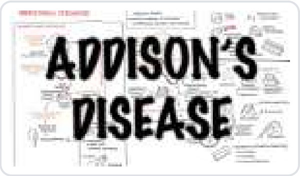
Clinical Knowledge Summaries 👍 12 👎 9

Addison's disease

Suggest link

Report broken link

Media



Addison's disease

Armando Hasudungan - YouTube 👍 3 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **61.2%**

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A 28-year-old woman is referred to the endocrinology department with abnormal thyroid function tests and neck pain. She has a past medical history of bipolar affecting disorder and takes lithium. She smokes five cigarettes daily. She is unemployed. She gave birth to her first child six weeks ago and has recently recovered from a coryzal illness. She is not breastfeeding.

On examination, there is a tender swelling in her neck. She is sweaty, mildly tremulous and tachycardia (heart rate 104 beats per minute). Examination of her eyes is unremarkable.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)
TSH	0.1 miU/L	(0.2 - 5.5)
Free T4	34 pmol/L	(0 - 24.5)

Additional tests:

Thyroid peroxidase antibodies	negative
Anti TSH receptor antibodies	negative

A radioactive iodine uptake scan reveals globally reduced uptake of iodine-131.

What is the likely diagnosis?

- ○ De Quervain's thyroiditis ✕
- ○ Factitious hyperthyroidism ✕
- ○ Grave's disease ✕
- ○ Lithium associated thyroiditis ✕
- ○ Post-partum thyroiditis ✕

Submit answer

Reference ranges ∨

Score: **61.2%**

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50 -

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A 28-year-old woman is referred to the endocrinology department with abnormal thyroid function tests and neck pain. She has a past medical history of bipolar affecting disorder and takes lithium. She smokes five cigarettes daily. She is unemployed. She gave birth to her first child six weeks ago and has recently recovered from a coryzal illness. She is not breastfeeding.

On examination, there is a tender swelling in her neck. She is sweaty, mildly tremulous and tachycardia (heart rate 104 beats per minute). Examination of her eyes is unremarkable.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)
TSH	0.1 miU/L	(0.2 - 5.5)
Free T4	34 pmol/L	(0 - 24.5)

Additional tests:

Thyroid peroxidase antibodies	negative
Anti TSH receptor antibodies	negative

A radioactive iodine uptake scan reveals globally reduced uptake of iodine-131.

What is the likely diagnosis?

De Quervain's thyroiditis	75%
Factitious hyperthyroidism	2%
Grave's disease	1%
Lithium associated thyroiditis	5%
Post-partum thyroiditis	18%

De Quervain's thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

Important for me Less important

De Quervain's thyroiditis is the correct answer. This woman presents with painful swelling in her neck (a likely goitre in the context of hyperthyroidism) post-coryzal illness with raised inflammatory markers (CRP), in association with globally reduced uptake of iodine-131. These findings are consistent with viral sub-acute thyroiditis.

Factitious hyperthyroidism is incorrect. Although she has a history of depression (and this is a risk factor for thyroid hormone misuse), this would not explain the raised CRP or neck pain.

Grave's disease is incorrect. This is associated with increased uptake of iodine-131 on a radioactive iodine scan.

Lithium associated thyroiditis is incorrect. This is classically 'painless' thyroiditis.

Post-partum thyroiditis is incorrect. While she is post-partum and this is a plausible differential, this condition is not associated with neck pain or raised inflammatory markers, making this diagnosis unlikely.

👍 👎 Discuss (4) Improve

Next question

Subacute (De Quervain's) thyroiditis

Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;

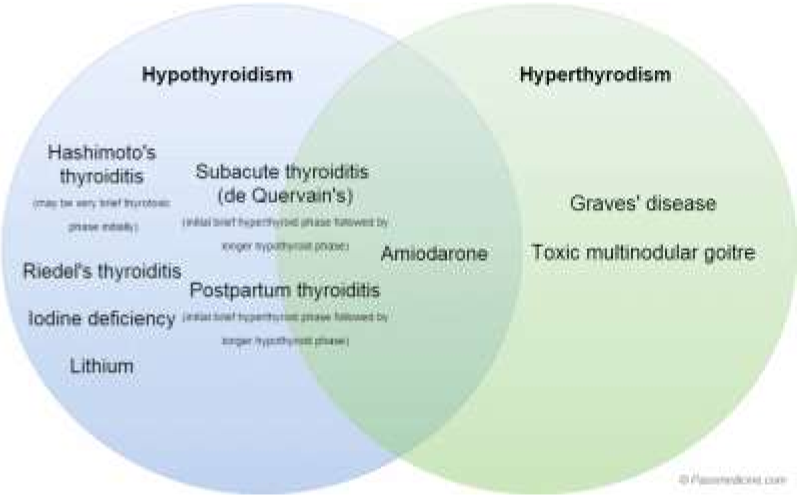
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations

- thyroid scintigraphy: globally reduced uptake of iodine-131

Management

- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Riedel's thyroiditis
Iodine deficiency
Lithium
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **62%**

1	✓
2	✓
3	✓
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45	✓
46	✓
47	✗
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50	✓

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Question 51 of 342

A 42-year-old man is referred from his GP to the outpatient department with a loss of libido and impotence. He has a stressful job as a pharmaceutical representative and has a five-year pack year history. He consumes four pints of beer per night.

Blood tests reveal a prolactin of 3340 mU/l (<360), normal thyroid function tests, MCV 100 fl, testosterone low.

Which of the following would be the most appropriate investigation?

- ○ Visual field testing ×
- ○ Urine cortisol ×
- ○ Pituitary dynamic function tests ×
- ○ MRI pituitary ×
- ○ Anterior pituitary function testing ×

Submit answer

Reference ranges

Score: **62%**

1 ✓
2 ✓
3 ✓

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50	✓
51	-

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A 42-year-old man is referred from his GP to the outpatient department with a loss of libido and impotence. He has a stressful job as a pharmaceutical representative and has a five-year pack year history. He consumes four pints of beer per night.

Blood tests reveal a prolactin of 3340 mU/l (<360), normal thyroid function tests, MCV 100 fl, testosterone low.

Which of the following would be the most appropriate investigation?

	Visual field testing	4%
	Urine cortisol	2%
	Pituitary dynamic function tests	15%
🟩	MRI pituitary	71%
🟥	Anterior pituitary function testing	9%

This patient has a prolactin result of over 3340 mU/l suggesting the most likely diagnosis is a prolactinoma. As the result is below 6000 mU/l would be suggestive of microprolactinoma as opposed to a macroprolactinoma. The most appropriate investigation would therefore be an MRI of the pituitary. On a side note, his raised MCV reinforces his history that he consumes too much alcohol.

👍 👎 Discuss (7) Improve

Next question

Prolactin and galactorrhoea ★

Prolactin is secreted by the anterior pituitary gland with release being controlled by a wide variety of physiological factors. Dopamine acts as the primary prolactin releasing inhibitory factor and hence dopamine agonists such as bromocriptine may be used to control galactorrhoea. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia

Features of excess prolactin

- men: impotence, loss of libido, galactorrhoea
- women: amenorrhoea, galactorrhoea

Causes of raised prolactin

- prolactinoma
- pregnancy
- oestrogens
- physiological: stress, exercise, sleep
- acromegaly: 1/3 of patients
- polycystic ovarian syndrome
- primary hypothyroidism (due to thyrotrophin releasing hormone (TRH) stimulating prolactin release)

Drug causes of raised prolactin

- metoclopramide, domperidone
- phenothiazines
- haloperidol
- very rare: SSRIs, opioids

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 11 👎 5

Galactorrhoea review

Suggest link Report broken link

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **60.8%**

1 ✓
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Question 52 of 342

A 42 year-old man presents to his GP with a 3 month history of increasing anxiety. On further questioning, he has lost 6 kg of weight over the past 2 months and has been experiencing increased bowel movements and diarrhoea.

Blood tests are performed and reveal:

Hb	14.2 g/dL	
Platelets	210 * 10^9/l	
WBC	6.9 * 10^9/l	
Thyroid stimulating hormone (TSH)	0.08 mu/l	
Free thyroxine (T4)	17.4 pmol/l	
Total triiodothyronine (T3)	13.4 nmol/l	Normal range (4.0-8.3 nmol/l)

What is the most appropriate treatment?

- ○ Reassurance ×
- ○ Carbimazole ×
- ○ Radio-iodine ×
- ○ Surgery ×
- ○ Propranolol ×

Submit answer

Reference ranges

Score: **60.8%**

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Free thyroxine (T4)	17.4 pmol/l	
Total triiodothyronine (T3)	13.4 nmol/l	Normal range (4.0-8.3 nmol/l)

What is the most appropriate treatment?

Reassurance	2%
[✓ Carbimazole]	77%
Radio-iodine	3%
Surgery	1%
Propranolol	17%

The diagnosis in this scenario is triiodothyronine thyrotoxicosis. A small subset of those patients experiencing thyrotoxicosis (roughly 5%) have isolated triiodothyronine thyrotoxicosis. As with other types of thyrotoxicosis, carbimazole is the main initial treatment for the condition.

👍 👎 Discuss (4) Improve

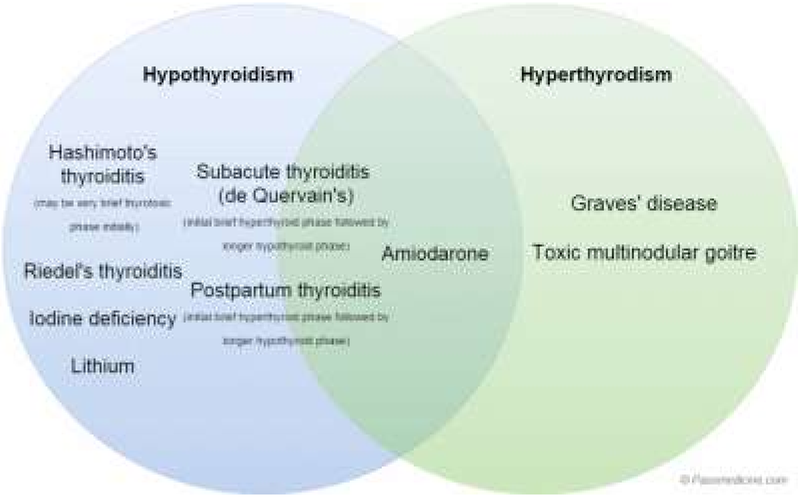
Next question

Thyrotoxicosis: causes and investigation

Graves' disease accounts for around 50-60% of cases of thyrotoxicosis.

Causes
- Graves' disease
- toxic nodular goitre
- acute phase of subacute (de Quervain's) thyroiditis
- acute phase of post-partum thyroiditis
- acute phase of Hashimoto's thyroiditis (later results in hypothyroidism)
- amiodarone therapy
- contrast
  - rare, more commonly occurs in elderly patients with pre-existing thyroid disease (e.g. multinodular goitre, Graves')
  - patients with existing thyrotoxicosis should not receive iodinated contrast medium
  - administration of iodinated contrast results in a large iodine load to the thyroid → hyperthyroidism developing over 2-12 weeks

Investigation
- TSH down, T4 and T3 up
- thyroid autoantibodies
- other investigations are not routinely done but includes isotope scanning


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 2 4

Hyperthyroidism guidelines

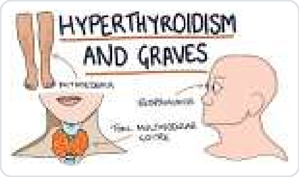
Royal College of Physicians 👍 7 👎 3

2017 Thyrotoxicosis â€“ investigation and management

Suggest link

Report broken link

Media


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 2 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube 👍 0 👎 0

Report broken media

Score: **61.5%**

1 ✓

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52	✓

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A 34-year-old man is admitted after being found at home covered in his vomit and excrement. He is a known alcoholic and has not been seen for two weeks.

On examination, he is drowsy and complaining of generalised aches and pains.

An ECG shows prolonged PR interval and prolonged QTc of 620ms.

On examination JVP is not visible, his mucus membranes are dry, and his eyes are sunken. His chest is clear, his heart sounds normal, and his abdomen soft and non-tender. There is moisture damage to his buttocks.

CT head is unremarkable.

Blood tests are completed and results are detailed below:

Hb	114 g/L	Male: (135-180) Female: (115 - 160)
Platelets	98 * 10^9/L	(150 - 400)
WBC	10 * 10^9/L	(4.0 - 11.0)

Na^+	131 mmol/L	(135 - 145)
K^+	2.2 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	190 µmol/L	(55 - 120)

Calcium	1.9 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
Magnesium	0.4 mmol/L	(0.7-1.0)

What is the priority in the management of this man's electrolyte disturbances?

- ○ Replace magnesium intravenously ×
- ○ Replace potassium intravenously ×
- ○ Start insulin-dex infusion ×
- ○ Start oral magnesium, potassium, phosphate, and calcium ×
- ○ Start telemetry ×

Submit answer

Reference ranges ∨

Score: **61.5%**

1	✓
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6	✓
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Question 53 of 342

A 34-year-old man is admitted after being found at home covered in his vomit and excrement. He is a known alcoholic and has not been seen for two weeks.

On examination, he is drowsy and complaining of generalised aches and pains.

An ECG shows prolonged PR interval and prolonged QTc of 620ms.

On examination JVP is not visible, his mucus membranes are dry, and his eyes are sunken. His chest is clear, his heart sounds normal, and his abdomen soft and non-tender. There is moisture damage to his buttocks.

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Blood tests are completed and results are detailed below:

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Calcium	1.9 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
Magnesium	0.4 mmol/L	(0.7-1.0)

What is the priority in the management of this man's electrolyte disturbances?

🟩	Replace magnesium intravenously	65%
🟥	Replace potassium intravenously	21%
	Start insulin-dex infusion	1%
	Start oral magnesium, potassium, phosphate, and calcium	5%
	Start telemetry	8%

Replace magnesium before correcting hypokalaemia. Hypomagnesemia prevents potassium absorption

Important for me Less important

In this question, the patient has multiple electrolyte abnormalities due to poor oral intake over the preceding 2 weeks.

Intravenous magnesium replacement would be recommended in this case due to the severity of magnesium deficiency. Severe hypomagnesaemia is defined as levels <0.4 mmol/L.

Although we do need to replace **potassium intravenously**, hypomagnesemia prevents potassium absorption, so we need to correct this before supplementing potassium.

Dex-saline can be used as part of the treatment of hyperkalaemia. It does not necessarily need to be used in hypokalaemia.

Start oral magnesium, potassium, phosphate, and calcium: this man has severe hypomagnesaemia (<0.4 mmol/L) and severe hypokalaemia (<2.5 mmol/L). He requires intravenous replacement and oral supplementation is not appropriate.

Telemetry would be required, but it is not the main priority here, electrolyte replacement should not be delayed in this case whilst awaiting telemetry.

👍 👎 Discuss (7) Improve

Next question

Hypokalaemia ★

Potassium and hydrogen can be thought of as competitors. Hyperkalaemia tends to be associated with acidosis because as potassium levels rise fewer hydrogen ions can enter the cells

Hypokalaemia with alkalosis

- vomiting
- thiazide and loop diuretics
- Cushing's syndrome
- Conn's syndrome (primary hyperaldosteronism)

Hypokalaemia with acidosis

- diarrhoea
- renal tubular acidosis
- acetazolamide
- partially treated diabetic ketoacidosis

Magnesium deficiency may also cause hypokalaemia. In such cases, normalizing the potassium level may be difficult until the magnesium deficiency has been corrected

+ Q 123

Next question

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Media

Hypokalemia - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **60.4%**

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A 52-year-old patient is referred to the endocrinology clinic due to excessive sweating, which occurs over several months, and was concerned when the GP was unable to reassure him. On further questioning he has persistent sweating which is out of keeping with his activities, and sometimes develops headaches. He has found this to be a worsening problem over the last months. He has found that he has low energy as well. On examination he has a large face and tongue. He has a past medical history of hypertension and type 2 diabetes.

What is the most appropriate first-line investigation

○ Serum growth hormone ×

○ Serum IGF-1 ×

○ Visual field testing ×

○ Oral glucose tolerance test measuring serum glucose ×

○ Pituitary MRI ×

Submit answer

Reference ranges

Score: **60.4%**

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Back to top

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What is the most appropriate first-line investigation

Serum growth hormone	2%
Serum IGF-1	88%
Visual field testing	1%
Oral glucose tolerance test measuring serum glucose	8%
Pituitary MRI	1%

Serum IGF-1 levels are now the first-line test for acromegaly

Important for me Less important

The most appropriate first-line investigation for this patient is **serum IGF-1**. The patient's symptoms and physical examination findings are suggestive of acromegaly, which is caused by excessive secretion of growth hormone (GH) from the pituitary gland. Acromegaly can lead to hypertension, type 2 diabetes, and other complications. Serum IGF-1 (insulin-like growth factor 1) levels are a reliable indicator of GH activity and are more stable than serum GH levels throughout the day. An elevated serum IGF-1 level would support the diagnosis of acromegaly.

Serum growth hormone measurement is not the best first-line investigation because GH secretion is pulsatile, with peaks and troughs throughout the day. This can lead to false-negative results if the blood sample is taken during a period when GH levels are low. Serum IGF-1 levels are less variable and provide a more accurate assessment of overall GH activity.

Visual field testing could be useful in assessing potential compression of the optic chiasm by a pituitary tumour causing acromegaly; however, it is not an appropriate first-line diagnostic test for confirming the presence of acromegaly itself. It would be more relevant once biochemical evidence has been obtained to support the diagnosis.

An **oral glucose tolerance test measuring serum glucose** may be helpful in evaluating a patient's diabetes status but does not directly address the suspicion of acromegaly. In patients with confirmed acromegaly, an oral glucose tolerance test measuring serum growth hormone may be used to assess disease activity, as normal individuals should have suppressed GH levels after glucose ingestion, while those with acromegaly will not suppress their GH adequately.

A **pituitary MRI** could potentially identify a pituitary adenoma as the cause of acromegaly; however, it is not the best first-line investigation. Biochemical confirmation of GH excess should be obtained before performing imaging studies. Once a diagnosis of acromegaly has been established through elevated serum IGF-1 levels and/or an abnormal oral glucose tolerance test measuring serum growth hormone, a pituitary MRI would then be appropriate to identify the underlying cause and guide further management.

👍 👎 Discuss (2) Improve

Next question

Acromegaly: investigations ★

Growth hormone (GH) levels vary during the day and are therefore not diagnostic.

Serum IGF-1 levels have now overtaken the oral glucose tolerance test (OGTT) with serial GH measurements as the first-line test. The OGTT test is recommended to confirm the diagnosis if IGF-1 levels are raised.

The Endocrine Society guidelines suggest the following:

> *1.1 We recommend measurement of IGF-1 levels in patients with typical clinical manifestations of acromegaly, especially those with acral and facial features.*
> ...
> *1.5 In patients with elevated or equivocal serum IGF-1 levels, we recommend confirmation of the diagnosis by finding lack of suppression of GH to < 1 μg/L following documented hyperglycemia during an oral glucose load.*

Serum IGF-1 may also be used to monitor disease

Oral glucose tolerance test
- in normal patients GH is suppressed to < 2 mu/L with hyperglycaemia
- in acromegaly there is no suppression of GH
- may also demonstrate impaired glucose tolerance which is associated with acromegaly

A pituitary MRI may demonstrate a pituitary tumour.

+ Q 123

Next question

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 14 👎 3

2014 Acromegaly guidelines

Suggest link

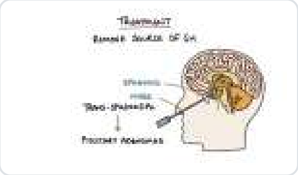
Report broken link

Media

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 1 👎 0



Understanding Acromegaly

Zero To Finals - YouTube 👍 1 👎 0

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A 19-year-old with type 1 diabetes presents to the Emergency Department feeling unwell. She states she has had vomiting and diarrhoea for 2 days and has not been taking her full insulin doses as she has been off her food. Her capillary glucose is 37 mmol/l and there are 4+ ketones on urinalysis.

An arterial blood gas is performed and the results are as follows:

pH	7.12
pO2	13 kPa
pCO2	3.5 kPa
HCO3	13
Na	129 mmol/l
K	6.1 mmol/l

Which of the following is the most appropriate initial management?

- ○ IV 0.9% NaCl bolus
- ○ IV 10 units actrapid + 50ml 50% dextrose
- ○ IV 8.4% sodium bicarbonate
- ○ Empirical IV antibiotics
- ○ Insulin sliding scale

Submit answer

Reference ranges

Score: **61.1%**

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Question 55 of 342 ✓

A 19-year-old with type 1 diabetes presents to the Emergency Department feeling unwell. She states she has had vomiting and diarrhoea for 2 days and has not been taking her full insulin doses as she has been off her food. Her capillary glucose is 37 mmol/l and there are 4+ ketones on urinalysis.

An arterial blood gas is performed and the results are as follows:

pH	7.12
pO2	13 kPa
pCO2	3.5 kPa
HCO3	13
Na	129 mmol/l
K	6.1 mmol/l

Which of the following is the most appropriate initial management?

IV 0.9% NaCl bolus	94%
IV 10 units actrapid + 50ml 50% dextrose	4%
IV 8.4% sodium bicarbonate	1%
Empirical IV antibiotics	0%
Insulin sliding scale	1%

This is a classical presentation of diabetic ketoacidosis. While precise protocols vary, the key principals are initial fluid resuscitation with normal saline prior to starting an IV insulin infusion, and careful potassium replacement.

Low sodium is often seen and is a pseudohyponatraemia secondary to the high serum glucose.

Serum potassium derangements are common and need careful management. Potassium is driven into cells by insulin. Serum potassium levels are therefore often high on presentation while blood insulin levels are depleted. Despite this, total body potassium is low due to fluid losses and requires careful monitoring and replacement during treatment.

Source:
http://www.diabetes.org.uk/Documents/About%20Us/What%20we%20say/Management-of-DKA-241013.pdf

👍 👎 Discuss (4) Improve

Next question

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration

- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids

  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following

commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

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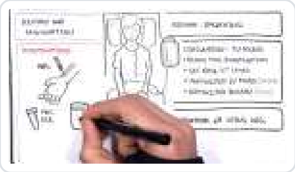
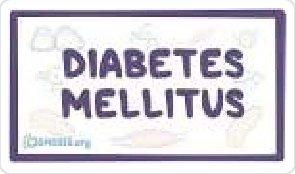
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2


DIABETES MELLITUS
Osmosis.org

Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **61.8%**

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A 46-year-old woman presents with polyuria. She has also noticed a swelling over the front of her neck and has been having headaches, sweating, and palpitations. Observations are as follows: heart rate 115 beats per minute, blood pressure 125/85 mmHg, respiratory rate 14 breaths per minute, SpO2 99% (on air), temperature 37.1°C.

Blood results are as follows:

Calcium	2.9 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
PTH	4.8 pmol/L	(1.6 - 6.9)
Thyroid stimulating hormone (TSH)	4.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	14.2 pmol/L	(9.0 - 18)

What investigation is required?

○ Radioactive iodine uptake scan ×
○ Serum calcitonin ×
○ Serum thyroglobulin ×
○ Thyroid peroxidase antibodies ×
○ Thyroid stimulating hormone receptor antibodies ×

Submit answer

Reference ranges

Score: **61.8%**

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Question 56 of 342 ✗

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PTH	4.8 pmol/L	(1.6 - 6.9)
Thyroid stimulating hormone (TSH)	4.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	14.2 pmol/L	(9.0 - 18)

What investigation is required?

	Radioactive iodine uptake scan	13%
🟩	Serum calcitonin	68%
	Serum thyroglobulin	8%
	Thyroid peroxidase antibodies	3%
🟥	Thyroid stimulating hormone receptor antibodies	8%

MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer

Important for me Less important

Serum calcitonin is correct. The patient most likely has multiple endocrine neoplasia (MEN) type 2. The presence of hypercalcemia with an 'inappropriately normal' PTH is suggestive of primary hyperparathyroidism. The patient also likely has a phaeochromocytoma due to the classic triad of episodic headache, sweating, and tachycardia. The neck swelling is concerning for medullary thyroid cancer which should be investigated with a thyroid ultrasound and serum calcitonin.

Radioactive iodine uptake scan is incorrect. Medullary thyroid cancer cells do not absorb iodine, and therefore radioiodine scans are not used to diagnose this type of cancer.

Serum thyroglobulin is incorrect. Papillary and follicular thyroid cancer release thyroglobulin which can therefore be used as a tumour marker for these types of cancer. However medullary cell cancer does not produce thyroglobulin. Medullary cell cancer cells produce calcitonin which can therefore be used as a tumour marker.

Thyroid peroxidase antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

Thyroid stimulating hormone receptor antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

👍 👎 Discuss (4) Improve

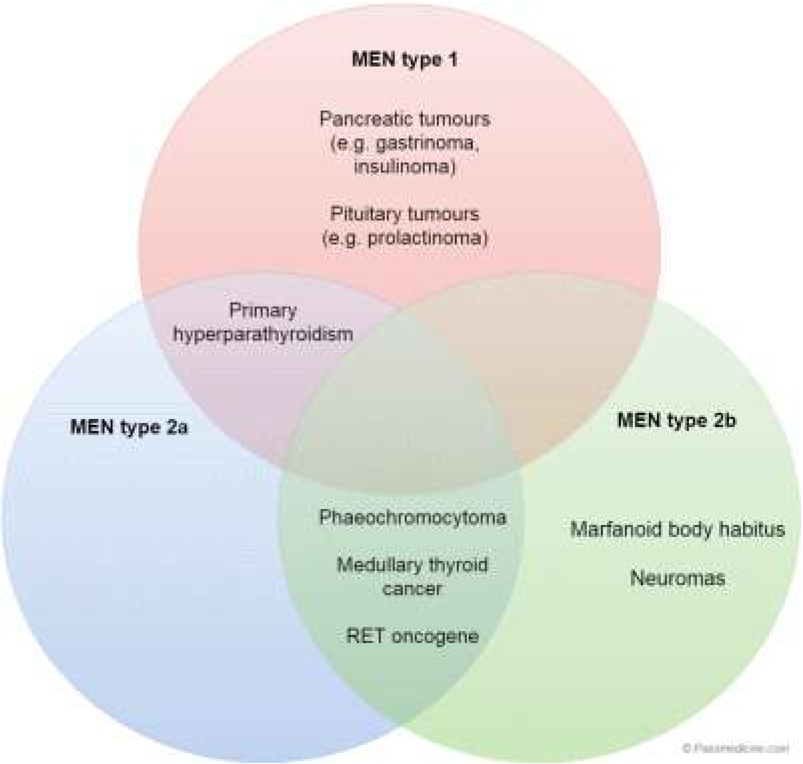
Next question

Multiple endocrine neoplasia

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	MEN type IIa	MEN type IIb
3 **P's**	Medullary thyroid cancer (70%)	Medullary thyroid cancer

MEN type I	**MEN type IIa**	**MEN type IIb**
Parathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas

Venn diagram showing the different types of MEN and their associated features

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Media

PANCREATIC
NEUROENDOCRINE
NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **60.7%**

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A 40-year-old female presented to Endocrinology Clinic with a 3-month history of weight gain, fatigue and headaches. Over the last 3 weeks, she has also experienced galactorrhoea and reduced libido. She was diagnosed with type 2 diabetes and hypertension 1 month ago and is on diet control for both. She is not currently on any regular medications. On examination, there was evidence of hirsutism and acne, a cervical fat pad, striae on her abdomen and proximal myopathy. Areas of hyperpigmentation were noted on her mucous membrane and palmar creases.

Which of the following investigations will reveal the diagnosis?

- ○ Low dose dexamethasone suppression test ×
- ○ Prolactin levels ×
- ○ Urinary cortisol ×
- ○ CT brain ×
- ○ MRI pituitary ×

Submit answer

Reference ranges

Score: **60.7%**

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Question 57 of 342 ✓

A 40-year-old female presented to Endocrinology Clinic with a 3-month history of weight gain, fatigue and headaches. Over the last 3 weeks, she has also experienced galactorrhoea and reduced libido. She was diagnosed with type 2 diabetes and hypertension 1 month ago and is on diet control for both. She is not currently on any regular medications. On examination, there was evidence of hirsutism and acne, a cervical fat pad, striae on her abdomen and proximal myopathy. Areas of hyperpigmentation were noted on her mucous membrane and palmar creases.

Which of the following investigations will reveal the diagnosis?

Low dose dexamethasone suppression test	39%
Prolactin levels	6%
Urinary cortisol	9%
CT brain	1%
[✓ MRI pituitary]	46%

The history suggests Cushing's syndrome, but the occurrence of galactorrhoea and reduced libido brings to attention the possibility of hyperprolactinaemia, and headaches may indicate an intracranial pathology. The diagnosis of a secreting pituitary tumour causing a raised level of prolactin and ACTH (causing hyperpigmentation) and hence, cortisol, should be suspected.

Low dose dexamethasone suppression test and 24-hour urinary cortisol will aid confirming the presence of Cushing's syndrome. Raised prolactin levels will confirm hyperprolactinaemia, however, it is the MRI pituitary that will lead to the diagnosis of a pituitary tumour.

Pituitary adenoma ★

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:
- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due to excess prolactin)
- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
  - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
    - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).
    - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
    - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone) and neuromodulators like pasireotide
- transsphenoidal surgery
    - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
    - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
    - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
    - indicated for residual or recurrent tumours post-surgery

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 20 👎 11

Pituitary tumours review

Suggest link Report broken link

Score: **61.4%**

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Question 58 of 342

A 35-year-old woman presents to the emergency department with headaches. These have been happening intermittently for the last 3 months but this one is particularly painful. They are associated with sweating and palpitations. She has found that headaches are often triggered after drinking coffee.

Neurological examination is normal. Observations are heart rate 119/min, blood pressure 186/108mmHg, respiratory rate 18/min, oxygen saturation 99%, temperature 37.3°C.

What is the most appropriate management?

- ○ Glyceryl trinitrate intravenous infusion ✕
- ○ Labetalol intravenous bolus injection ✕
- ○ Labetalol intravenous infusion ✕
- ○ Phenoxybenzamine intravenous infusion ✕
- ○ Phenoxybenzamine orally ✕

Submit answer

Reference ranges

Score: **61.4%**

1 ✓

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Neurological examination is normal. Observations are heart rate 119/min, blood pressure 186/108mmHg, respiratory rate 18/min, oxygen saturation 99%, temperature 37.3°C.

What is the most appropriate management?

	Glyceryl trinitrate intravenous infusion	2%
	Labetalol intravenous bolus injection	6%
	Labetalol intravenous infusion	11%
🟥	Phenoxybenzamine intravenous infusion	33%
🟩	Phenoxybenzamine orally	48%

PHaeochromocytoma - give **PH**enoxybenzamine before beta-blockers

Important for me Less important

The combination of headache, tachycardia, and diaphoresis alongside significant hypertension is suggestive of phaeochromocytoma. This is additionally supported by the intermittent nature of the symptoms. There are several recognised triggers for 'attacks', including dietary factors such as cheese, tomatoes, and coffee.

Both alpha-blockers and beta-blockers are used in the management of phaeochromocytoma. Alpha-blockers oppose catecholamine-induced vasoconstriction. An alpha-blockade must be performed first with the alpha-blocker phenoxybenzamine. This medication only has oral forms available and licenced in the BNF and therefore **phenoxybenzamine orally** is the correct answer. If the beta-receptors are blocked first this leads to a loss of beta-mediated vasodilation and the subsequent unopposed alpha-adrenergic receptor stimulation can precipitate extreme hypertension.

A **glyceryl trinitrate intravenous infusion** can be used for treating significant hypertension. In phaeochromocytoma, hypertension is primarily driven by the activation of the sympathetic nervous system through the release of catecholamines acting on alpha receptors. Therefore, the most appropriate and effective way to counter this is through an alpha-blocker.

Labetalol is a beta-blocker. Although beta-blockers play a role in the management of phaeochromocytoma, it is important to give alpha-blockers first as mentioned above. Therefore, both **labetalol intravenous bolus injection** and **labetalol intravenous infusion** are incorrect.

Phenoxybenzamine intravenous infusion is incorrect, only oral forms of this medication are available and licenced in the BNF.

👍 👎 Discuss (4) Improve

Next question

Phaeochromocytoma

Phaeochromocytoma is a rare catecholamine secreting tumour. About 10% are familial and may be associated with MEN type II, neurofibromatosis and von Hippel-Lindau syndrome

Basics
- bilateral in 10%
- malignant in 10%

- extra-adrenal in 10% (most common site = organ of Zuckerkandl, adjacent to the bifurcation of the aorta)

Features are typically episodic
- hypertension (around 90% of cases, may be sustained)
- headaches
- palpitations
- sweating
- anxiety

Tests
- 24 hr urinary collection of metanephrines (sensitivity 97%*)
- this has replaced a 24 hr urinary collection of catecholamines (sensitivity 86%)

Surgery is the definitive management. The patient must first however be stabilized with medical management:
- alpha-blocker (e.g. phenoxybenzamine), given before a
- beta-blocker (e.g. propranolol)

*BMJ 2012; 344 doi: http://dx.doi.org/10.1136/bmj.e1042 (Published 20 February 2012)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PHEOCHROMOCYTOMA

Phaeochromocytoma

Townsend Teaching - YouTube 👍 3 👎 0

Report broken media

Score: **60.3%**

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Question 59 of 342

A 75-year-old lady presents to the acute medical unit. She is referred by her GP who is concerned that she has been exposed to the chicken pox virus. Her grandson currently has a generalised vesicular rash and she had been in contact with him earlier that day. Her past medical history includes heart failure and giant cell arteritis. Her current medications include prednisolone, ramipril and bisoprolol. She has been taking prednisolone 40mg for the past two weeks.

How will you manage this patient?

- ○ Intravenous ganciclovir ✕
- ○ Intravenous aciclovir ✕
- ○ Oral aciclovir ✕
- ○ Check Varicella Zoster antibodies ✕
- ○ Varicella Zoster immunoglobulin ✕

Submit answer

Reference ranges

Score: **60.3%**

1 ✓
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Back to top

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How will you manage this patient?

	Intravenous ganciclovir	1%
	Intravenous aciclovir	3%
	Oral aciclovir	9%
🟩	Check Varicella Zoster antibodies	57%
🟥	Varicella Zoster immunoglobulin	29%

Any patient who is taking 40 mg or more of prednisiolone for more than 7 days or who is taking 20 mg or more of prednisolone for more than 14 days is classed as immunocompromised

Important for me Less important

Varicella Zoster immunoglobulin (VZIG) prophylaxis is recommended for individuals who fulfil all of the following three criteria:

- 1. Significant exposure to chickenpox or herpes zoster
- 2. A clinical condition that increases the risk of severe varicella; this includes immunosuppressed patients, neonates and pregnant women
- 3. No antibodies to Varicella Zoster (VZ) virus

Any patient who is taking 40 mg or more of prednisolone for more than 7 days or who is taking 20 mg or more of prednisolone for more than 14 days is classed as immunocompromised.

This patient is therefore immunosuppressed and has been exposed to the VZ virus. Antibodies to VZ virus should, therefore, be checked so that VZ immunoglobulin can be administered if the patient is seronegative.

Primary varicella zoster virus (VZV) infection or chickenpox is typically a benign illness in immunocompetent individuals. However, the risk of life-threatening complications, such as disseminated VZV and pneumonitis, is greater in immunosuppressed individuals, with significant rates of mortality.

👍 👎 Discuss (8) Improve

Next question

Corticosteroids

Corticosteroids are amongst the most commonly prescribed therapies in clinical practice. They are used both systemically (oral or intravenous) or locally (skin creams, inhalers, eye drops, intra-articular). They augment and in some cases replace the natural glucocorticoid and mineralocorticoid activity of endogenous steroids.

The relative glucocorticoid and mineralocorticoid activity of commonly used steroids is shown below:

Minimal glucocorticoid activity, very high mineralocorticoid activity,	**Glucocorticoid activity, high mineralocorticoid activity,**	**Predominant glucocorticoid activity, low mineralocorticoid activity**	**Very high glucocorti activity, m mineraloc activity**
Fludrocortisone	Hydrocortisone	Prednisolone	Dexameth Betmethas

Side-effects

The side effects of corticosteroids are numerous and represent the single greatest limitation on their usage. Side-effects are more common with systemic and prolonged therapy.

Glucocorticoid side-effects

- endocrine
    - impaired glucose regulation
    - increased appetite/weight gain
    - hirsutism
    - hyperlipidaemia
- Cushing's syndrome
    - moon face
    - buffalo hump
    - striae
- musculoskeletal
    - osteoporosis
    - proximal myopathy
    - avascular necrosis of the femoral head
- immunosuppression
    - increased susceptibility to severe infection
    - reactivation of tuberculosis
- psychiatric
    - insomnia
    - mania
    - depression
    - psychosis
- gastrointestinal
    - peptic ulceration

    - acute pancreatitis
- ophthalmic
    - glaucoma
    - cataracts
- suppression of growth in children
- intracranial hypertension
- neutrophilia

Mineralocorticoid side-effects
- fluid retention
- hypertension

Selected points on the use of corticosteroids:
- patients on long-term steroids should have their doses doubled during intercurrent illness
- longer-term systemic corticosteroids suppress the natural production of endogenous steroids. They should therefore not be withdrawn abruptly, as this may precipitate an Addisonian crisis
- the BNF suggests gradual withdrawal of systemic corticosteroids if patients have:
    - received more than 40mg prednisolone daily for more than one week
    - received more than 3 weeks of treatment
    - recently received repeated courses

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **59.3%**

1	✓
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A 56-year-old man with a history of type 2 diabetes managed with Humalog mix 30 and metformin 1g BD comes to the clinic for review. HbA1c is currently 57. He has troublesome hypoglycaemia episodes in the late afternoon and early mornings and wants to know what to do about it. On examination his blood pressure is 132/82 mmHg, his pulse is 72 beats per minute and regular. His body mass index is 32 kg/m².

Investigations

Na^+	139 mmol/l
K^+	4.9 mmol/l
Urea	5.1 mmol/l
Creatinine	94 μmol/l
HbA1c	57 mmol/mol

Which of the following is the most appropriate next step in his management?

- ○ Continue current regimen, eat a snack mid afternoon and before bed time ×
- ○ Reduce the dose of metformin ×
- ○ Reduce the dose of mixed insulin ×
- ○ Switch to a basal bolus regimen ×
- ○ Switch to Humalog mix 20 ×

Submit answer

Reference ranges

Score: **59.3%**

1	✓
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Investigations

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K^+	4.9 mmol/l
Urea	5.1 mmol/l
Creatinine	94 μmol/l
HbA1c	57 mmol/mol

Which of the following is the most appropriate next step in his management?

Continue current regimen, eat a snack mid afternoon and before bed time	8%
Reduce the dose of metformin	1%
Reduce the dose of mixed insulin	22%
Switch to a basal bolus regimen	63%
Switch to Humalog mix 20	6%

This person's HbA1c is above target at 57 mmol/mol or 7.4%. In spite of this, he is still suffering from hypoglycaemia episodes as a result of delivering his insulin as a twice a day regimen. Moving him to basal bolus will allow splitting up of the short acting component of his insulin requirements, and therefore reduce the risk of hypoglycaemia at the end of each dosing period.

Reducing the dose of his mixed insulin may drive a further rise in his HbA1c, as would reducing the dose of metformin. Switching to Humalog mix 20 will increase the proportion of long-acting insulin and worsen the risk of hypoglycaemia. Eating a snack will drive weight gain and worsen his HbA1c.

👍 👎 Discuss (7) Improve

Next question

Insulin therapy: side-effects ★

Hypoglycaemia

- patients should be taught the signs of hypoglycaemia: sweating, anxiety, blurred vision, confusion, aggression
- conscious patients should take 10-20g of a short-acting carbohydrate (e.g. a glass of Lucozade or non-diet drink, three or more glucose tablets, glucose gel)
- every person treated with insulin should have a glucagon kit for emergencies where the patient is not able to orally ingest a short-acting carbohydrate
- patients who have frequent hypoglycaemic episodes may develop reduced awareness. If this develops then allowing glycaemic control to slip for a period of time may restore their awareness
- beta-blockers reduce hypoglycaemic awareness

Lipodystrophy

- typically presents as atrophy/lumps of subcutaneous fat
- can be prevented by rotating the injection site
- may cause erractic insulin absorption

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **60%**

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60	✓

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A 46-year-old man presents to the Endocrinology clinic after a referral from his GP for asymptomatic hypercalcaemia, which was recently discovered on some routine blood tests. His past medical history is significant only for hypertension, for which he is taking amlodipine, ramipril and chlorthalidone.

Selected investigation results are shown below:

Na^+	141 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Calcium	2.85 mmol/L	(2.2 - 2.6)
Urea	6.6 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
Parathyroid hormone	5.5 pmol/L	(1.6 - 6.9)

Urinary calcium	30mg/24h	(100 - 300)

What is the most likely explanation for this patient's hypercalcaemia?

○ Chlorthalidone use ×

○ Familial hypocalciuric hypercalcaemia ×

○ Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion ×

○ Primary hyperparathyroidism ×

○ Secondary hyperparathyroidism ×

Submit answer

Reference ranges

Score: **60%**

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Selected investigation results are shown below:

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Urea	6.6 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
Parathyroid hormone	5.5 pmol/L	(1.6 - 6.9)

Urinary calcium	30mg/24h	(100 - 300)

What is the most likely explanation for this patient's hypercalcaemia?

Chlorthalidone use	22%
[✓ Familial hypocalciuric hypercalcaemia]	63%
Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion	1%
Primary hyperparathyroidism	13%
Secondary hyperparathyroidism	0%

Low urinary calcium in the presence of hypercalcaemia is suggestive of either familial hypocalciuric hypercalcaemia or thiazide diuretic use

Important for me Less important

The correct answer is **familial hypocalciuric hypercalcaemia**. This patient has hypercalcemia with low urinary calcium, which narrows the diagnosis down to familial hypocalciuric hypercalcemia (FHH) or thiazide diuretic usage (other causes of hypercalcemia tend to cause elevated urinary calcium). The diagnosis of FHH is clinched by the inappropriately normal parathyroid hormone. This is because, in FHH, loss of function mutations in the CASR calcium-sensing receptor decrease sensitivity to calcium, meaning PTH remains unsuppressed at higher-than-normal serum calcium levels. Hypocalciuria results from a loss of CASR-mediated negative feedback of tubular reabsorption/ excretion of calcium.

Chlorthalidone use is incorrect, as this would give hypocalciuric hypercalcaemia with a suppressed PTH.

Hypercalcaemia of malignancy with ectopic parathyroid hormone-related protein secretion is incorrect. This would classically lead to hypercalciuria. In addition, serum PTH may be suppressed rather than inappropriately normal in this scenario, as the PTHrp peptide is detected by a different assay from the conventional PTH assay.

Primary hyperparathyroidism is incorrect. Although this could explain the hypercalcaemia and inappropriately normal PTH, this would lead to hypercalciuria rather than hypocalciuria.

Secondary hyperparathyroidism is incorrect. Here, PTH is released in response to hypocalcaemia, often in the context of renal disease. The elevated serum calcium and normal renal function seen in this patient do not support this diagnosis.

👍 👎 Discuss (8) Improve

Next question

Familial benign hypocalciuric hypercalcaemia

Familial benign hypocalciuric hypercalcaemia is a rare autosomal dominant disorder characterised by asymptomatic hypercalcaemia. It is due to a defect in the calcium-sensing receptor and a decreased sensitivity to increases in extracellular calcium.

The parathyroid hormone level is often not suppressed, as would be expected in all non-hyperparathyrodism related cases of hypercalcaemia. This is due to the decreased sensitivity to increases in extracellular calcium.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

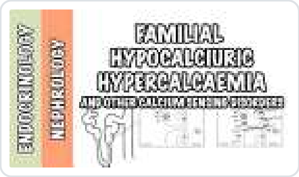
Links

Oxford Academic Website 👍 2 👎 0

Urine Calcium: Laboratory Measurement and Clinical Utility

Suggest link Report broken link

Media


ENDOCRINOLOGY
NEPHROLOGY
FAMILIAL HYPOCALCIURIC HYPERCALCAEMIA
AND OTHER CALCIUM SENSING DISORDERS

Familial Hypocalciuric Hypercalcemia + other calcium sensing disorders

Armando Hasudungan - YouTube 👍 7 👎 1

Report broken media

Score: **60.7%**

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Question 62 of 342

A 42-year-old lady comes to see you in outpatients. Incidentally, you notice that her TSH was < 0.1 mU/l on a recent blood test requested by the GP. Her only past medical history is thyroid cancer which has been resected and her only medication levothyroxine 100mcg per day.

She is otherwise asymptomatic. What is the best course of action?

- ○ Stop levothyroxine ✕
- ○ Continue at 100mcg per day ✕
- ○ Repeat thyroid function in 6 weeks ✕
- ○ Change to liothyronine equivalent dose ✕
- ○ Reduce the levothyroxine as she is over treated ✕

Submit answer

Reference ranges

Score: **60.7%**

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Question 62 of 342 ✗

A 42-year-old lady comes to see you in outpatients. Incidentally, you notice that her TSH was < 0.1 mU/l on a recent blood test requested by the GP. Her only past medical history is thyroid cancer which has been resected and her only medication levothyroxine 100mcg per day.

She is otherwise asymptomatic. What is the best course of action?

	Stop levothyroxine	3%
🟩	Continue at 100mcg per day	25%
	Repeat thyroid function in 6 weeks	21%
	Change to liothyronine equivalent dose	2%
🟥	Reduce the levothyroxine as she is over treated	49%

As TSH is a growth factor for many thyroid cancers it is routinely suppressed with levothyroxine. TSH and thyroglobulin should be monitored in patients with a history of thyroid cancer and decisions on the level required are usually specialist led.

High and intermediate risk patients should have their TSH suppressed bellow 0.1 mU/l and low-risk patients TSH should be 0.1-0.5 mU/l.

As she is asymptomatic and her TSH is adequately suppressed this lady should be maintained on her current dose pending specialist review.

British Thyroid Association, guidelines for the management of thyroid cancer (2014) and The American Thyroid Associations Guidelines (2009).

Discuss (9) | Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer
- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	**Notes**
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei

Type	Notes
	• Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

Suggest link

Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0

Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **59.7%**

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Question 63 of 342

A 29-year-old woman presents to the ENT clinic with hoarseness. She has found herself to be hoarse for three weeks and despite drinking plenty of fluids finds herself unable to improve. She was referred to the ENT clinic after a phone consultation with her GP. On examination she has a goitre which is not painful. On further questioning she has no symptoms consistent with an overactive or underactive thyroid. She also feels well in herself. She has no problem speaking apart from the hoarseness, and is not struggling to catch her breath. What is the most appropriate course of action?

- ○ Immediate hospital admission under endocrinology ×
- ○ Two-week wait appointment with an endocrinologist ×
- ○ Routine appointment with an endocrinologist ×
- ○ Reassure and discharge back to GP ×
- ○ Arrange for bronchoscopy ×

Submit answer

Reference ranges

Score: **59.7%**

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← Question 63 of 342 ✕ ⚑ →

A 29-year-old woman presents to the ENT clinic with hoarseness. She has found herself to be hoarse for three weeks and despite drinking plenty of fluids finds herself unable to improve. She was referred to the ENT clinic after a phone consultation with her GP. On examination she has a goitre which is not painful. On further questioning she has no symptoms consistent with an overactive or underactive thyroid. She also feels well in herself. She has no problem speaking apart from the hoarseness, and is not struggling to catch her breath. What is the most appropriate course of action?

	Immediate hospital admission under endocrinology	28%
🟩	Two-week wait appointment with an endocrinologist	47%
🟥	Routine appointment with an endocrinologist	10%
	Reassure and discharge back to GP	4%
	Arrange for bronchoscopy	11%

Goitre with unexplained hoarseness should be referred urgently

Important for me Less important

The combination of thyroid swelling with hoarseness or change in voice which is unexplained needs urgent referral to endocrinology for likely thyroid cancer. Thyroid nodules in a child, thyroid mass with cervical lymphadenopathy or a rapidly enlarging and painless thyroid mass also need a two-week wait referral for suspect thyroid cancer. A patient with a thyroid mass and stridor may need immediate admission. Patients with a thyroid swelling and abnormal thyroid function tests, or sudden pain in a thyroid lump, can be referred non-urgently.

👍 👎 Discuss (11) Improve

Next question

Thyroid cancer ★

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	Percentage	
Papillary	70%	Often young females - excellent prognosis
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Anaplastic carcinoma	- Most common in elderly females - Local invasion is a common feature - Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

Suggest link Report broken link

Media

THYROID
CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0

Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **58.7%**

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Question 64 of 342

You are asked to review a 43-year-old man in theatre recovery who has developed a fever and tachycardia post-operatively. He is previously fit and well, does not smoke and drinks alcohol only occasionally. He had fallen the previous night and suffered a distal radius fracture and has just undergone a open reduction and internal fixation under general anaesthetic. During anaesthesia he received 4mg ondansetron and 8mg dexamethasone for post-operative nausea and 10mg morphine for pain. He denies feeling unwell and has no symptoms suggestive of intercurrent infection.

On examination his heart rate is 130 beats/min and irregular, his blood pressure is 135/74 mmHg and his temperature is 39.4ºC. His chest is clear to auscultation, his abdomen soft and non-tender and there is no rash or meningism. His right forearm is in plaster, but is not particularly painful and his fingers are warm and have normal sensation.

Hb	130 g/l
Platelets	460 * 10^9/l
WBC	10.5 * 10^9/l
Na$^+$	138 mmol/l
K$^+$	4.1 mmol/l
Urea	5.1 mmol/l
Creatinine	95 µmol/l
C-reactive protein	1 mg/L
Thyroid stimulating hormone	<0.02 mIU/L
Cortisol	45 µg/dL

What is the most appropriate initial treatment?

○ Carbimazole ✕

- ○ Hydrocortisone ✕
- ○ Propranolol ✕
- ○ Broad spectrum antibiotics ✕
- ○ Crystalloid infusion ✕

Submit answer

Reference ranges

Score: **58.7%**

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Na^+	138 mmol/l
K^+	4.1 mmol/l
Urea	5.1 mmol/l
Creatinine	95 µmol/l
C-reactive protein	1 mg/L
Thyroid stimulating hormone	<0.02 mIU/L
Cortisol	45 µg/dL

What is the most appropriate initial treatment?

Carbimazole 5%

Hydrocortisone	28%
Propranolol	60%
Broad spectrum antibiotics	2%
Crystalloid infusion	4%

The diagnosis here is thyrotoxicosis as a presenting feature of hyperthyroidism. Infection is unlikely given the normal clinical examination and normal CRP. During initial treatment of thyrotoxicosis it is important to treat hypoadrenalism first - if present - in order to not precipitate a addisonian crisis. However, this patient has no features in the history to suggest pre-existing Addisons disease, he has normal electrolytes and the suppressed cortisol can be explained by the peri-operative use of dexamethasone. Initial treatment of thyrotoxicosis should focus on sympathetic storm suppression using beta blockade. Anti-thyroid medications - i.e. carbimazole - take up to six weeks to take full effect and are not useful in the acute scenario.

Discuss (14) Improve

Next question

Thyrotoxicosis: causes and investigation

Graves' disease accounts for around 50-60% of cases of thyrotoxicosis.

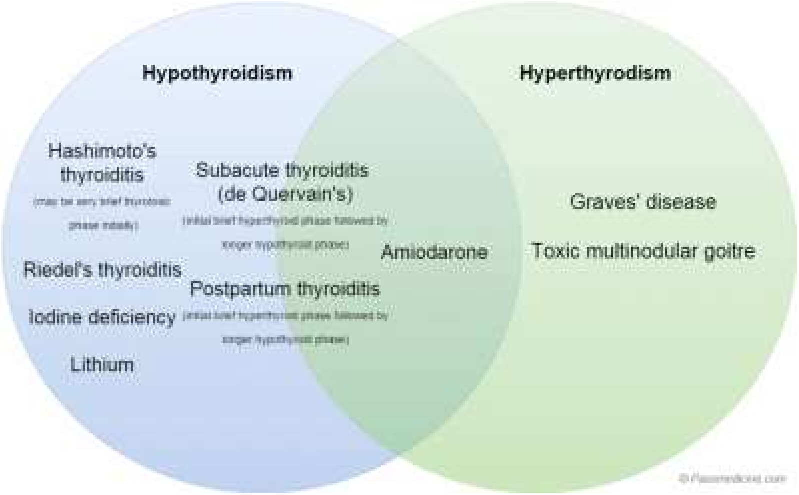
Causes

- Graves' disease
- toxic nodular goitre
- acute phase of subacute (de Quervain's) thyroiditis
- acute phase of post-partum thyroiditis
- acute phase of Hashimoto's thyroiditis (later results in hypothyroidism)
- amiodarone therapy
- contrast

    - rare, more commonly occurs in elderly patients with pre-existing thyroid disease (e.g. multinodular goitre, Graves')
    - patients with existing thyrotoxicosis should not receive iodinated contrast medium
    - administration of iodinated contrast results in a large iodine load to the thyroid → hyperthyroidism developing over 2-12 weeks

Investigation

- TSH down, T4 and T3 up
- thyroid autoantibodies
- other investigations are not routinely done but includes isotope scanning


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Riedel's thyroiditis
Postpartum thyroiditis
Iodine deficiency
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Lithium
Graves' disease
Toxic multinodular goitre
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 2 👎 4

Hyperthyroidism guidelines

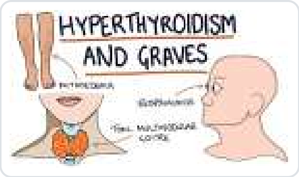
Royal College of Physicians 👍 7 👎 3

2017 Thyrotoxicosis â€“ investigation and management

Suggest link

Report broken link

Media


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 2 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube 👍 0 👎 0

Report broken media

Score: **57.8%**

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Question 65 of 342

A 55-year-old man was seen in the Emergency Department after a fainting episode. He describes a history of fatigue and nausea. His past medical history includes type 2 diabetes mellitus and HIV infection and he admits that he has not been compliant with any medications, including his anti-retroviral therapy.

On examination, his pulse was 65 beats per minute and regular, blood pressure 90/62 mmHg and respiratory rate 26 breaths per minute.

Investigations:

Haemoglobin	14.0 g/dL (13.0-18.0)
White cell count	4 x 10^9/L (4-11)
Platelets	150 x 10^9/L (150-400)
Sodium	130 mmol/L (135-145)
Potassium	5.8 mmol/L (3.5-5.0)
Creatinine	80µmol/L (60-110)
Glucose	4.0 mmol/L (4.0-7.8)

What is the most appropriate next step management step?

- ○ Salbutamol nebuliser ×
- ○ Broad-spectrum antibiotics ×
- ○ Sliding scale insulin infusion ×
- ○ Intravenous hydrocortisone ×
- ○ Restart anti-retroviral medications ×

Submit answer

Reference ranges

Score: **57.8%**

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Investigations:

Haemoglobin	14.0 g/dL (13.0-18.0)
White cell count	4 x 10^9/L (4-11)
Platelets	150 x 10^9/L (150-400)
Sodium	130 mmol/L (135-145)
Potassium	5.8 mmol/L (3.5-5.0)
Creatinine	80µmol/L (60-110)
Glucose	4.0 mmol/L (4.0-7.8)

What is the most appropriate next step management step?

	Salbutamol nebuliser	4%
	Broad-spectrum antibiotics	2%
	Sliding scale insulin infusion	2%
🟩	Intravenous hydrocortisone	85%
	Restart anti-retroviral medications	7%

Adrenal insufficiency affects approximately 10% of patients with HIV, commonly due to cytomegalovirus (CMV)-related necrotising adrenalitis. It is the failure of the adaptive immune system seen in HIV infection, and particularly AIDS, that increases susceptibility to CMV infection, and thus risk of CMV-associated adrenal failure.

The immediate management of hypoadrenalism involves rapid steroid replacement; fluid resuscitation would also be a priority.

There is no firm evidence here to suggest bacterial infection, and the hyperkalaemia should resolve with appropriate management of the hypoadrenalism.

👍 👎 Discuss (1) Improve

Next question

Addisonian crisis

Causes

- sepsis or surgery causing an acute exacerbation of chronic insufficiency (e.g. Addison's, hypopituitarism)
- adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcaemia)
- steroid withdrawal (most common precipitant)
- inadequate 'sick day rule' management or intercurrent illness (commonly gastroenteritis) in patients with underlying Addison's disease

Signs and symptoms

- confusion, syncope, lethargy, convulsions
- hypotension, tachycardia, hypothermia
- hyponatraemia, hyperkalaemia, hypoglycaemia
- abdominal pain or cramps

Management

- hydrocortisone 100 mg IM or IV followed by 200 mg per 24 hours as a continuous infusion or 50 mg IV or IM every 6 hours (taper

according to response as the patient becomes stable)
- 1 litre of normal saline infused over 30–60 minutes, then a further 4–6 litres over the first 24 hours if required; include dextrose if hypoglycaemic
- no fludrocortisone is required acutely as high-dose cortisol exerts a weak mineralocorticoid effect
- once stable, convert to oral replacement after approximately 24 hours, reducing to maintenance dosing over 3–4 days
- identify and treat the precipitating cause (e.g. infection)
- educate patients (and carers) about 'sick day rules' and encourage use of steroid alert cards or bracelets to prevent future crises

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 8 2

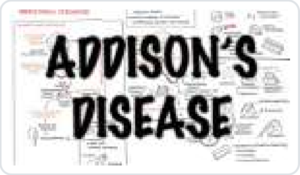
2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 6 👎 0



Addison's disease

Armando Hasudungan - YouTube 👍 4 👎 0

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Question 66 of 342

A 50-year-old man who drives a heavy goods vehicle comes to the diabetes clinic for review. Current medication for diabetes includes metformin 1g BD and gliclazide 160mg BD. His blood sugars have steadily increased over the past few months and his most recent HbA1c is 72 mmol/mol. He has increased in weight by 5kg over the past 12 weeks which he puts down to work, (driving his lorry for excessive periods). On examination his blood pressure is 155/88 mmHg, pulse is 75 beats per minute and regular. Abdomen is soft and non-tender, body mass index is 37 kg/m².

Investigations:

Na^+	140 mmol/l
K^+	5.0 mmol/l
Urea	7.1 mmol/l
Creatinine	110 µmol/l

Which of the following is the most appropriate next step?

- ○ Add basal insulin ×
- ○ Add mixed insulin ×
- ○ Add pioglitazone ×
- ○ Switch gliclazide to linagliptin ×
- ○ Switch gliclazide to liraglutide ×

Submit answer

Reference ranges ⌄

Score: **58.5%**

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A 50-year-old man who drives a heavy goods vehicle comes to the diabetes clinic for review. Current medication for diabetes includes metformin 1g BD and gliclazide 160mg BD. His blood sugars have steadily increased over the past few months and his most recent HbA1c is 72 mmol/mol. He has increased in weight by 5kg over the past 12 weeks which he puts down to work, (driving his lorry for excessive periods). On examination his blood pressure is 155/88 mmHg, pulse is 75 beats per minute and regular. Abdomen is soft and non-tender, body mass index is 37 kg/m².

Investigations:

Na^+	140 mmol/l
K^+	5.0 mmol/l
Urea	7.1 mmol/l
Creatinine	110 µmol/l

Which of the following is the most appropriate next step?

	Add basal insulin	4%
	Add mixed insulin	2%
	Add pioglitazone	11%
	Switch gliclazide to linagliptin	16%
✓	Switch gliclazide to liraglutide	68%

A number of factors come into play with respect to management of this patient's blood glucose. His occupation as a lorry driver precludes medication which may significantly increase his risk of hypoglycaemia. This effectively rules out insulin initiation unless he is prepared to accept any impact on his job. Insulin would also promote further weight gain and salt and water retention, and is therefore less desirable as an option. Pioglitazone promotes weight gain and fluid retention, so again is not really an option.

This leaves us with the two switching scenarios. Switching to liraglutide is preferred, because in trials GLP-1 receptor agonists have been shown to reduce HbA1c by similar levels to basal insulin, without increasing the risk of hypoglycaemia, and promote approximately 3% weight loss over a 6 month period. DPPIV inhibitors such as linagliptin are potentially less effective in reducing HbA1c than sulphonylureas and linagliptin is therefore not an option here.

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Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice
- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish

- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

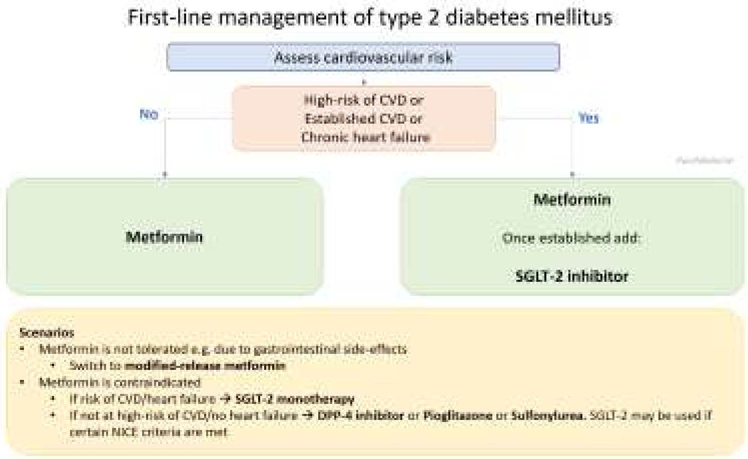
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

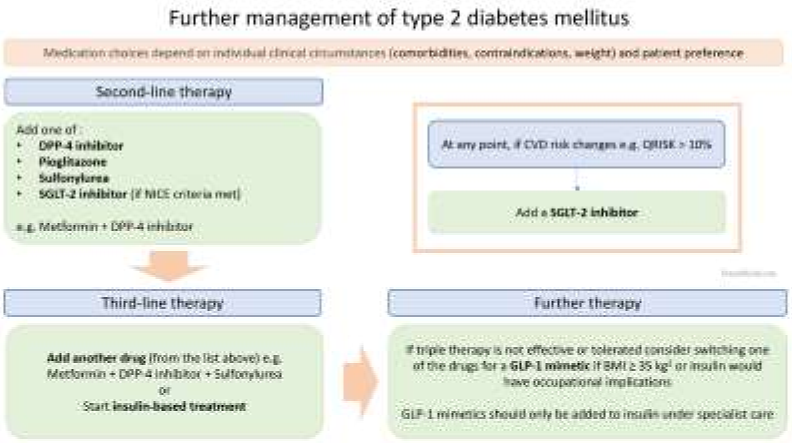
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is

indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

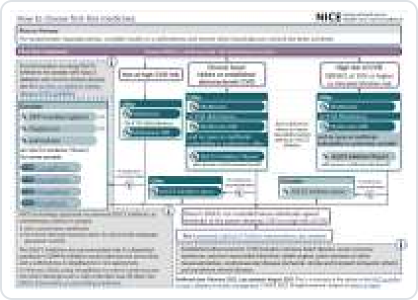
GLP-1 mimetics should only be added to insulin under specialist care

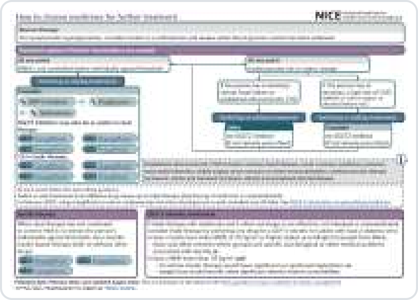
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

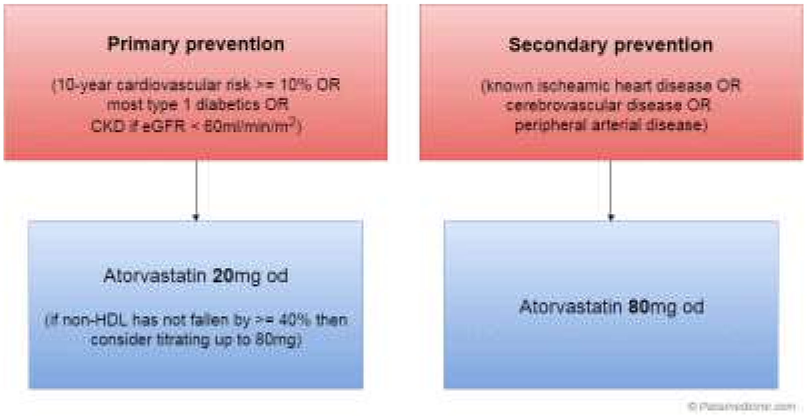
Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be

offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
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Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

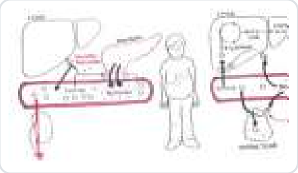
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

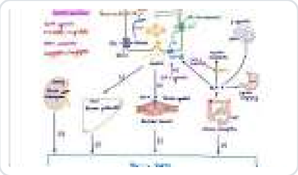
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

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Question 67 of 342

A 34-year-old woman presents with a history of infertility, amenorrhoea, and galactorrhoea over the past 6 months. She also reports headaches and visual disturbances, including blurry vision and difficulty seeing objects in her peripheral field.

On examination, her blood pressure is 125/80 mmHg, heart rate is 82 bpm, and BMI is 28 kg/m². Neurological examination reveals bitemporal hemianopia. Routine blood tests are as follows:

Prolactin	3500 mU/L	50 - 450 mU/L
TSH	0.5 mU/L	0.4 - 4.0 mU/L
Free T4	13 pmol/L	12 - 22 pmol/L

MRI of the brain shows a pituitary macroadenoma (1.5 cm) with mild compression of the optic chiasm.

What is the best next step in the management of this patient?

- ○ Cabergoline ✕
- ○ High-dose corticosteroids ✕
- ○ Octreotide ✕
- ○ Pegvisomant ✕
- ○ Transsphenoidal surgical resection ✕

Submit answer

Reference ranges

Score: **59.1%**

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On examination, her blood pressure is 125/80 mmHg, heart rate is 82 bpm, and BMI is 28 kg/m². Neurological examination reveals bitemporal hemianopia. Routine blood tests are as follows:

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TSH	0.5 mU/L	0.4 - 4.0 mU/L
Free T4	13 pmol/L	12 - 22 pmol/L

MRI of the brain shows a pituitary macroadenoma (1.5 cm) with mild compression of the optic chiasm.

What is the best next step in the management of this patient?

Cabergoline	47%
High-dose corticosteroids	0%
Octreotide	1%
Pegvisomant	0%
Transsphenoidal surgical resection	52%

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

Important for me Less important

This patient’s amenorrhoea, infertility, galactorrhoea, mild visual disturbances, and markedly elevated prolactin (3500 mU/L) with a 1.5 cm adenoma strongly suggest a prolactinoma. Dopamine agonists like **cabergoline** and bromocriptine are the first-line treatment, even for large tumours, as they effectively lower prolactin levels and induce significant tumour shrinkage, alleviating both endocrine and compressive symptoms. Most macroprolactinomas respond well to medical therapy alone, and even in cases where further intervention is needed, initial dopamine agonist therapy helps optimise management.

This patient’s symptoms are best explained by a prolactinoma, and **High-dose corticosteroids** have no role in its management. Steroids are primarily used in pituitary apoplexy, a condition caused by haemorrhage or infarction of the pituitary gland, which presents acutely with sudden-onset severe headache, visual loss, ophthalmoplegia, and adrenal insufficiency. In contrast, this patient has gradual-onset symptoms, an elevated prolactin level, and MRI findings consistent with a macroadenoma, making dopamine agonists the appropriate first-line treatment.

Octreotide, a somatostatin analogue, is primarily used in the treatment of acromegaly and certain neuroendocrine tumour, as it inhibits growth hormone secretion. However, it has no significant effect on prolactinomas and is not a recommended first-line treatment. While somatostatin analogue may have a minor role in resistant prolactinomas, dopamine agonists remain the most effective therapy. In this patient, the combination of infertility, amenorrhoea, galactorrhoea, and a markedly elevated prolactin level (3500 mU/L) strongly supports a diagnosis of a prolactinoma rather than acromegaly.

This patient’s symptoms, along with elevated prolactin levels and a pituitary macroadenoma, are indicative of a prolactinoma, not acromegaly. **Pegvisomant** is used to treat acromegaly by blocking the actions of excess growth hormone at its receptor, thereby reducing the effects of growth hormone excess. However, it has no role in treating prolactinomas, as the pathophysiology and management differ significantly.

Transsphenoidal surgical resection is not the first-line treatment for this patient. Her markedly elevated prolactin level (3500 mU/L) strongly suggests a prolactinoma rather than a non-functioning pituitary adenoma (NFPA), which typically causes only mild-to-moderate hyperprolactinaemia (usually < 2000 mU/L) due to stalk effect. While NFPAs can present with compressive symptoms such as visual field defects, this patient's biochemical profile indicates a prolactin-secreting tumour. Surgery is generally reserved for cases where dopamine agonists fail to control tumour growth or alleviate symptoms, or if there is severe, progressive visual impairment. Given her mild optic chiasmal compression and the high likelihood of response to medical therapy, dopamine agonists remain the preferred first-line treatment.

👍 👎 Discuss Improve

Next question

Prolactinoma ★

Prolactinomas are a type of pituitary adenoma, a benign tumour of the pituitary gland.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is >1cm)
- **hormonal status** (a secretory/functioning adenoma produces and excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin.

Features
- excess prolactin in women
    - amenorrhoea
    - infertility
    - galactorrhoea
    - osteoporosis
- excess prolactin in men
    - impotence

    - loss of libido
    - galactorrhoea
- other symptoms may be seen with macroadenomas
    - headache.
    - visual disturbances (classically, a bitemporal hemianopia (lateral visual fields) or upper temporal quadrantanopia)
    - symptoms and signs of hypopituitarism

Diagnosis

- MRI

Management

- in the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. cabergoline, bromocriptine) which inhibit the release of prolactin from the pituitary gland
- surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 5 👎 2

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Score: **59.7%**

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A 76 year old woman was admitted to hospital after presenting to the Emergency Department with shortness of breath, productive cough and palpitations. A chest x-ray demonstrated a left lower lobe pneumonia and ECG showed atrial fibrillation with a fast ventricular response. Initial management included intravenous antibiotics, intravenous fluids and oral digoxin loading.

Two days after admission, the patient's condition had significantly improved and she was able to start mobilising on the ward. The palpitations that she had been experiencing at presentation had also ceased. Following review by the Senior House Officer on the ward round, a repeat ECG was requested when demonstrated that the patient had cardioverted back to sinus rhythm. Digoxin therapy was subsequently held.

To investigate for an underlying cause of atrial fibrillation, thyroid function tests were added to blood tests from admission, with results as listed below.

Haemoglobin	125 g / dL
White cell count	13.7* 10^9/l
Neutrophils	11.9* 10^9/l
Platelets	351 * 10^9/l
Urea	4.6 mmol / L
Creatinine	130 micromol / L
Sodium	139 mmol / L
Potassium	3.6 mmol / L
C-reactive protein	105 mg / L
Thyroid stimulating hormone	0.25 microU / L
T4 free serum	14.1 pmol / L
T3 free serum	7.4 pmol / L

What is the most appropriate next investigation to assess deranged thyroid function tests?

- ○ Thyroid ultrasound ✕
- ○ Thyroid peroxidase antibody levels ✕
- ○ Repeat TFT in 6 weeks ✕
- ○ Thyroid scintiscanning ✕
- ○ Thyroglobulin antibody levels ✕

Submit answer

Reference ranges ⌵

Score: **59.7%**

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A 76 year old woman was admitted to hospital after presenting to the Emergency Department with shortness of breath, productive cough and palpitations. A chest x-ray demonstrated a left lower lobe pneumonia and ECG showed atrial fibrillation with a fast ventricular response. Initial management included intravenous antibiotics, intravenous fluids and oral digoxin loading.

Two days after admission, the patient's condition had significantly improved and she was able to start mobilising on the ward. The palpitations that she had been experiencing at presentation had also ceased. Following review by the Senior House Officer on the ward round, a repeat ECG was requested when demonstrated that the patient had cardioverted back to sinus rhythm. Digoxin therapy was subsequently held.

To investigate for an underlying cause of atrial fibrillation, thyroid function tests were added to blood tests from admission, with results as listed below.

Haemoglobin	125 g / dL
White cell count	13.7* 10^9/l
Neutrophils	11.9* 10^9/l
Platelets	351 * 10^9/l
Urea	4.6 mmol / L
Creatinine	130 micromol / L
Sodium	139 mmol / L
Potassium	3.6 mmol / L
C-reactive protein	105 mg / L
Thyroid stimulating hormone	0.25 microU / L
T4 free serum	14.1 pmol / L
T3 free serum	7.4 pmol / L

What is the most appropriate next investigation to assess deranged thyroid function tests?

	Thyroid ultrasound	7%
🟥	Thyroid peroxidase antibody levels	12%
🟩	Repeat TFT in 6 weeks	63%
	Thyroid scintiscanning	12%
	Thyroglobulin antibody levels	6%

Sick euthyroid is common in unwell, elderly patients and often needs no treatment

Important for me Less important

Any acute and severe illness may alter thyroid hormone deiodination through the effects of cytokines and result in various changes in levels of TSH, fT3 and fT4. Low TSH levels in hospitalised patients are three times more likely to be due to this effect than to hyperthyroidism.

It is therefore best to avoid thyroid function testing around the time of an acute illness unless there is good clinical evidence of a primary thyroid illness. If TFT remain deranged following recovery from acute illness then further investigation to assess for thyroid disease can be considered.

Discuss (11) Improve

Next question

Sick euthyroid syndrome

In sick euthyroid syndrome (now referred to as non-thyroidal illness) it is often said that everything (TSH, thyroxine and T3) is low. In the majority of cases however the TSH level is within the >normal range (inappropriately normal given the low thyroxine and T3).

Changes are reversible upon recovery from the systemic illness and hence no treatment is usually needed.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **58.8%**

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A 42-year-old woman presented to ambulatory care clinic with a three month history of increasing tiredness, lethargy, and poor concentration. She had lost weight because of a reduction in appetite due to nonspecific

abdominal pain. The pain was not altered by eating, movement, or related to having her bowels open. There was no associated vomiting or diarrhoea. She recently had to return early from holiday in Mexico because she felt so unwell. On examination, she was slim and tanned from her recent holiday. Her temperature was 36.9°C, pulse 89 beats per minute, blood pressure 90/60 mmHg, and respiratory rate 18 breaths per minute. Cardiovascular and respiratory examination was normal. Her abdomen was soft and non-tender.

Investigations:

Hb	128g/l (115-165)
WBC	10.2 * 10^9/l (4.0-11.0)
Na$^+$	128mmol/l (137-144)
K$^+$	5.6mmol/l (3.5-4.9)
Urea	10.8mmol/l (2.5-7.0)
Creatinine	98µmol/l (60-110)
Albumin	38g/l (37-49)
Alkaline phosphatase	126U/l (45-105)
ALT	112U/l (5-35)
Bilirubin	12µmol/l (1-22)
Alkaline phosphatase	126U/l (45-105)
Amylase	826U/l (60-180)
Ca$^+$	2.84mmol/l (2.20-2.60)

What is the next most appropriate investigation for diagnosis to be made?

○ Dexamethasone suppression test ✕

○ Short synacthen test ✕

○ Protein electrophoresis ✕

○ Thyroid function test ✕

○ Urine and plasma osmolality ✕

Submit answer

Reference ranges

Score: **58.8%**

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Investigations:

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Alkaline phosphatase	126U/l (45-105)
Amylase	826U/l (60-180)
Ca$^+$	2.84mmol/l (2.20-2.60)

What is the next most appropriate investigation for diagnosis to be made?

Dexamethasone suppression test	4%
Short synacthen test	85%
Protein electrophoresis	3%
Thyroid function test	4%
Urine and plasma osmolality	3%

Primary hypoadrenalism is diagnosed by a short synacthen test and a failure to increase cortisol levels to above 500nmol/L

Important for me Less important

The scenario depicts primary hypoadrenalism. The classic electrolyte disturbance in primary hypoadrenalism (Addison's disease) is hyponatraemia (in about 90%) and hyperkalaemia (about 65%). Hyperkalaemia occurs because of the loss of aldosterone production. Hyponatraemia is partly due to sodium depletion but also vasopressin levels are elevated, resulting in an increase in free water retention. Reversible changes to liver enzymes may also occur. Hypercalcaemia (6%) may also occur. This is partly due to a reduction in calcium removal by the kidney and an increase in mobilization of calcium from bone stores. Thyroid stimulating hormone (TSH) can be elevated in primary hypoadrenalism due to a loss of feedback on the pituitary gland.

There are no features of hypercortisolism to necessitate a dexamethasone suppression test. Urine and plasma osmolalities maybe useful when evaluating hyponatraemia of unknown cause but on their own not useful in making specific diagnosis. Multiple myeloma is unlikely in the above scenario.

👍 👎 Discuss (11) Improve

Next question

Addison's disease: investigations

In a patient with suspected Addison's disease the definite investigation is an ACTH stimulation test (short Synacthen test). Plasma cortisol is measured before and 30 minutes after giving Synacthen 250ug IM. Adrenal autoantibodies such as anti-21-hydroxylase may also be demonstrated.

If an ACTH stimulation test is not readily available (e.g. in primary care) then sending a 9 am serum cortisol can be useful:

- > 500 nmol/l makes Addison's very unlikely
- < 100 nmol/l is definitely abnormal
- 100-500 nmol/l should prompt a ACTH stimulation test to be performed

Associated electrolyte abnormalities are seen in around one-third of undiagnosed patients:

- hyperkalaemia
- hyponatraemia
- hypoglycaemia
- metabolic acidosis

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 14 👎 7

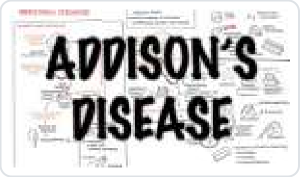
2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 2 👎 1



Addison's disease

Armando Hasudungan - YouTube 👍 2 👎 1

Report broken media

Score: **59.4%**

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A 65-year-old man attends with a hot swollen right toe. On examination there is swelling over the MTP joint of the right toe. There is reduced range of movement and it is hot, swollen and painful to touch. He has a past medical history of alcohol excess. He takes no regular medications.

An aspirate of the joint fluid is as follows:

Microscopy	Negatively birefringent crystals - needle shaped

The patient is intolerant of NSAIDs and colchicine. You therefore start a trial of prednisolone. Unfortunately the patient develops steroid induced psychosis.

What agent will you consider prescribing?

- ○ Canakinumab ×
- ○ Ibuprofen ×
- ○ Naproxen ×
- ○ Paracetamol ×
- ○ Allopurinol ×

Submit answer

Reference ranges

Score: **59.4%**

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Question 70 of 342 ✗

A 65-year-old man attends with a hot swollen right toe. On examination there is swelling over the MTP joint of the right toe. There is reduced range of movement and it is hot, swollen and painful to touch. He has a past medical history of alcohol excess. He takes no regular medications.

An aspirate of the joint fluid is as follows:

Microscopy	Negatively birefringent crystals - needle shaped

The patient is intolerant of NSAIDs and colchicine. You therefore start a trial of prednisolone. Unfortunately the patient develops steroid induced psychosis.

What agent will you consider prescribing?

🟩	Canakinumab	66%
	Ibuprofen	2%
🟥	Naproxen	6%
	Paracetamol	8%
	Allopurinol	17%

Canakinumab is a human monoclonal antibody that selectively inhibits interleukin-1 beta receptor binding. It can be used for treatment of acute gout has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them

Important for me Less important

Canakinumab is a human monoclonal antibody that selectively inhibits interleukin-1 beta receptor binding. It can be used for treatment of acute gout has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them.

The patient is intolerant of NSAIDs therefore ibuprofen and naproxen would be inappropriate.

Paracetamol is a simple analgesic and unlikely to control the patients pain. In addition, the inflammatory nature of gout warrants the use of a drug which reduces inflammation.

Allopurinol is indicated for recurrent gout. It should not be initiated in the acute setting as doing so can prolong the duration and severity of the gout.

👍 👎 Discuss (4) Improve

Next question

IL-1 ★

Interleukin 1 (IL-1) is a key mediator of the immune response. It is secreted mainly by macrophages and monocytes and acts as a costimulator of T cell and B cell proliferation.

Other effects include increasing the expression of adhesion molecules on the endothelium. By stimulating the release by the endothelium of vasoactive factors such as PAF, nitric oxide and prostacyclin it also causes vasodilation and increases vascular permeability. It is therefore one of the mediators of shock in sepsis. Along with IL-6 and TNF, it acts on the hypothalamus causing pyrexia.

IL-1 inhibitors are increasingly used in medicine. Examples include:

- anakinra
    - IL-1 receptor antagonist
    - used in the management of rheumatoid arthritis
- canakinumab
    - monoclonal antibody targeted at IL-1 beta

    - used systemic juvenile idiopathic arthritis and adult-onset Still's disease

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

IMMUNOLOGY
PHARMACOLOGY
INTERLEUKIN 1
(IL-1 CYTOKINE)

Interleukin 1 (IL1) physiology and IL1 antagonist pharmacology

Armando Hasudungan - YouTube 👍 2 👎 0

Report broken media

Score: **58.6%**

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← Question 71 of 342 →

A 65-year-old Muslim man with type II diabetes on metformin (500mg three times a day) comes to your endocrine clinic. He is about to start fasting between sunrise and sunset for Ramadan. He will typically eat a light meal before sunrise (Suhoor) and a large meal at sunset (Iftar).

How would you advise this gentleman in respect to his metformin prior to the large meal at sunset?

- ○ Change to a sulphonylurea ×
- ○ Stop metformin ×
- ○ Take 1.5g metformin before the large meal at sunset ×
- ○ Take 500mg metformin before the large meal at sunset ×
- ○ Take 1g metformin before the large meal at sunset ×

Submit answer

Reference ranges

Score: **58.6%**

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← Question 71 of 342 ✗ →

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How would you advise this gentleman in respect to his metformin prior to the large meal at sunset?

Change to a sulphonylurea	0%
Stop metformin	0%
Take 1.5g metformin before the large meal at sunset	9%
Take 500mg metformin before the large meal at sunset	6%
Take 1g metformin before the large meal at sunset	84%

This patient will still require metformin and therefore it is not appropriate to stop it.

The total daily dose of metformin should be split:

- One-third before sunrise (Suhoor) (500mg)
- Two-thirds after sunset (Iftar) (1g)

There is no need to change to a sulphonylurea if his blood glucose levels are controlled on metformin alone.

Discuss (4) Improve

Next question

Diabetes mellitus: Ramadan

We know that type 2 diabetes mellitus is more common in people of Asian ethnicity and a significant proportion of those patients in the UK will be Muslim. The BMJ published an excellent and comprehensive review of this issue in 2010[1].

It is important that we can give appropriate advice to Muslim patients to allow them safely observe their fast. This is particularly important from 2014 as Ramadan is due to fall in the long days of the summer months for several years henceforth.

Clearly it is a personal decision whether a patient decides to fast. It may however be worthwhile exploring the fact that people with chronic conditions are exempt from fasting or may be able to delay fasting to the shorter days of the winter months. It is however known that many Muslim patients with diabetes do not class themselves as having a chronic/serious condition which should exempt them from fasting. Around 79% of Muslim patients with type 2 diabetes mellitus fast Ramadan[2].There is an excellent patient information leaflet from Diabetes UK and the Muslim Council of Britain which explores these options in more detail.

If a patient with type 2 diabetes mellitus does decide to fast:
- they should try and and eat a meal containing long-acting carbohydrates prior to sunrise (Suhoor)
- patients should be given a blood glucose monitor to allow them to check their glucose levels, particularly if they feel unwell
- for patients taking metformin the expert consensus is that the dose should be split one-third before sunrise (Suhoor) and two-thirds after sunset (Iftar)
- expert consensus also recommends switching once-daily sulfonylureas to after sunset. For patients taking twice-daily preparations such as gliclazide it is recommended that a larger proportion of the dose is taken after after sunset
- no adjustment is needed for patients taking pioglitazone

1. Management of people with diabetes wanting to fast during Ramadan

BMJ 2010;340:c3053
2. Salti I et al. Results of the Epidemiology of Diabetes and Ramadan (EPIDIAR) study. Diabetes Care 2004;27:2306-11.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Diabetes Care 👍 22 👎 6

Recommendations for Management of Diabetes During Ramadan

Diabetes UK and Muslim Council 👍 25 👎 4

Patient information leaflet on the management of diabetes during Ramadan

Suggest link

Report broken link

Score: **57.7%**

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A 37-year-old female presents to the medical outpatient department with a progressive loss of libido. She attributes this to persistent diarrhoea, which she has noted over the last 6 months. She has also lost 16kg of weight and feels fatigued. She has noticed that her eyes have become grossly protuberant and she has double vision on looking towards either the right or left. She also experiences painful watering of her eyes.

On examination she has a marked tremor in both hands, her heart rate is irregularly irregular and she has marked exophthalmos. There is an audible bruit on auscultation of the thyroid gland.

Her laboratory investigations reveal:

Hb	130 g/l
MCV	77 fl
MCH	29 pg
WBC	7.4 * 10^9/l
Plt	430 * 10^9/l
TSH	0.03 mU/l (0.4 3.6 mU/l)
Total T4	302 nmol/l (68 174 nmol/l)

CT scan of the orbits reveals taut optic nerves and retro-orbital oedema.

Which of the following would be the most appropriate management of her eye condition?

○ Treatment with a block and replace regimen ×

○ Treatment with radioactive iodine ×

○ Treatment with IV methylprednisolone ×

○ Surgical removal of the thyroid gland ×

○ Orbital decompression surgery ×

Submit answer

Reference ranges

Score: **57.7%**

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Back to top

Question 72 of 342 ✓

A 37-year-old female presents to the medical outpatient department with a progressive loss of libido. She attributes this to persistent diarrhoea, which she has noted over the last 6 months. She has also lost 16kg of weight and feels fatigued. She has noticed that her eyes have become grossly protuberant and she has double vision on looking towards either the right or left. She also experiences painful watering of her eyes.

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CT scan of the orbits reveals taut optic nerves and retro-orbital oedema.

Which of the following would be the most appropriate management of her eye condition?

Treatment with a block and replace regimen	9%
Treatment with radioactive iodine	5%
Treatment with IV methylprednisolone	65%

Surgical removal of the thyroid gland	5%
Orbital decompression surgery	15%

The question aims to address the ophthalmopathy associated with Graves disease and tests the candidates understanding of the correct approach to a patient with painful and significant eye disease. The treatment of choice is the administration of systemic steroids to lessen the inflammation and provide symptomatic relief. Treatment of the underlying thyrotoxicosis is essential, but it will not directly result in an improvement in the ophthalmopathy.

Radioactive iodine therapy may worsen Graves ophthalmopathy and should not be the initial treatment option. Treatment-induced hypothyroidism must be avoided as this may also worsen the eye problems.

👍 👎 Discuss (7) Improve

Next question

Thyroid eye disease ★

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology
- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention
- smoking is the most important modifiable risk factor for the development of thyroid eye disease

- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features

- the patient may be eu-, hypo- or hyperthyroid at the time of presentation
- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management

- smoking cessation
- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery

Complications

Exposure keratopathy

- this is the most common complication of thyroid eye disease
- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy

- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour

vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia
- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)
- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):
- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes
- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy 👍 9 👎 8

Management of Graves' Orbitopathy

Suggest link

Report broken link

Score: **58.3%**

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Question 73 of 342

A 22-year-old female, who is known to have type 1 diabetes mellitus, presents with weight loss, anorexia and fatigue for the last six months.

Her diabetes was well controlled with soluble insulin three times daily and long acting insulin in the evening but during the last six months she noticed that her insulin requirement has generally decreased and on three occasions she had hypoglycaemic attacks.

During the same time period she had lost approximately 7 Kg in weight and had generally lost her appetite. She had also been amenorrhoeic over the last three months.

On examination, she is thin (BMI 18), with a pulse rate of 70 beats per minute and a blood pressure of 110/70 mmHg with a postural drop.

Investigations reveal:

Serum sodium	125mmol/L
Serum potassium	5.3mmol/L
Serum urea	7.4mmol/L
Serum creatinine	100 mol/L
Serum glucose	7.5mmol/L
HbA1c	6.0%
Serum free T4	7.5 pmol/L
Serum TSH	5.5 pmol/L
Serum oestradiol	70 pmol/L (130-850)
Serum LH	2.5 mU/L (2-10)
Serum FSH	2.2 mU/L (2-10)
Serum prolactin	400 mU/L (50-450)
Serum calcium	2.9 mmol/l
Serum phosphate	0.8 mmol/l

What is the most appropriate investigation for this patient?

- ○ Thyroid autoantibodies ×
- ○ PTH concentration ×
- ○ Short synacthen test ×
- ○ Pregnancy test ×
- ○ Random cortisol concentration ×

Submit answer

Reference ranges ⌄

Score: **58.3%**

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Dr. Jamil Alrajhi

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Serum prolactin	400 mU/L (50-450)
Serum calcium	2.9 mmol/l
Serum phosphate	0.8 mmol/l

What is the most appropriate investigation for this patient?

	Thyroid autoantibodies	5%
🟥	PTH concentration	9%
🟩	Short synacthen test	74%
	Pregnancy test	7%
	Random cortisol concentration	6%

This known type 1 diabetic female has developed Addisons disease on top of her diabetes. This explains the hypoglycaemic attacks, the decrease in her insulin requirement, the fatigue and weight loss.

Both type 1 DM and Addisons disease are features of Schmidt's disease (type 2 autoimmune polyendocrine syndrome) which is the diagnosis in this case.

The low T4, raised TSH, high calcium, low FSH, low LH, low oestradiol (hypogonadotrophic hypogonadism) are all features of Addisons disease.

The best investigation to diagnose Addisons disease is the short synacthen test.
It is of paramount importance when treating these patients is not to replace thyroxine before hydrocortisone because this will induce Addisonian crisis.

👍 | 👎 | 💬 Discuss (13) | Improve

Next question

Addisonian crisis ★

Causes

- sepsis or surgery causing an acute exacerbation of chronic insufficiency (e.g. Addison's, hypopituitarism)
- adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcaemia)
- steroid withdrawal (most common precipitant)
- inadequate 'sick day rule' management or intercurrent illness (commonly gastroenteritis) in patients with underlying Addison's disease

Signs and symptoms

- confusion, syncope, lethargy, convulsions
- hypotension, tachycardia, hypothermia
- hyponatraemia, hyperkalaemia, hypoglycaemia
- abdominal pain or cramps

Management

- hydrocortisone 100 mg IM or IV followed by 200 mg per 24 hours as a continuous infusion or 50 mg IV or IM every 6 hours (taper according to response as the patient becomes stable)
- 1 litre of normal saline infused over 30–60 minutes, then a further 4–6 litres over the first 24 hours if required; include dextrose if hypoglycaemic
- no fludrocortisone is required acutely as high-dose cortisol exerts a weak mineralocorticoid effect
- once stable, convert to oral replacement after approximately 24 hours, reducing to maintenance dosing over 3–4 days
- identify and treat the precipitating cause (e.g. infection)
- educate patients (and carers) about 'sick day rules' and encourage use of steroid alert cards or bracelets to prevent future crises

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 8 👎 2

2017 Adrenal insufficiency â€“ recognition and management

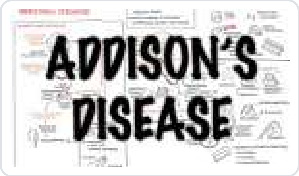
Suggest link

Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 6 👎 0



Addison's disease

Armando Hasudungan - YouTube 👍 4 👎 0

Report broken media

Score: **57.5%**

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Question 74 of 342 ✗

A 67-old-lady presents a week after a recent episode of *Campylobacter* gastroenteritis with increased thirst and passing urine. She estimates that she drinks 9-10 glasses of water or tea a day which is twice what she would normally have. In addition, her urine is clear in colour. She feels low in energy with easy fatigue but is able to continue her voluntary work at a local shop. She reports occasional cramps in her legs after standing for some time. She takes indapamide for hypertension but is otherwise well.

On examination, she appears euvolaemic with moist mucosa and no oedema. Her abdomen is soft with bowel sounds . She appears slow, overweight and lethargic. with a There are no neck masses or eye changes. Blood pressure is 105/90mmHg and heart rate is 67/min.

Na^+	138mmol/l
K^+	2.6 mmol/l
Urea	4.2 mmol/l
Creatinine	53 µmol/l
Calcium (corrected)	2.34 mmol/l (2.2-2.6)
TSH	3.2 mU/L (normal range 0.4-4.0)
Cortisol	400 nmol/l (normal range 129-450)
Water deprivation test	pending

What is the likeliest cause of these symptoms?

Addison's disease	8%
Hypothyroidism	6%
Syndrome of inappropriate ADH secretion	8%
Psychogenic polydipsia	27%

Hypokalaemia 51%

Hypokalaemia is a rare cause of polyuria and polydipsia

Important for me Less important

This lady has polydipsia and polyuria. Of the above options, only hypokalaemia and psychogenic polydipsia are recognised causes of these symptoms. Hyperthyroidism can rarely cause polydipsia but not hypothyroidism. Addison's disease can cause hypercalcaemia which can cause these symptoms but her calcium is normal. Psychogenic polydipsia can occur but this is usually a chronic problem. This lady has presented very soon after her gastroenteritis and has evidence of hypokalaemia making this a rare but the likeliest cause of her symptoms. Her indapamide is probably contributing to the persistent hypokalaemia and should be stopped.

👍 👎 Discuss (1) Improve

Next question

Hypokalaemia ★

Potassium and hydrogen can be thought of as competitors. Hyperkalaemia tends to be associated with acidosis because as potassium levels rise fewer hydrogen ions can enter the cells

Hypokalaemia with alkalosis
- vomiting
- thiazide and loop diuretics
- Cushing's syndrome
- Conn's syndrome (primary hyperaldosteronism)

Hypokalaemia with acidosis
- diarrhoea
- renal tubular acidosis

- acetazolamide
- partially treated diabetic ketoacidosis

Magnesium deficiency may also cause hypokalaemia. In such cases, normalizing the potassium level may be difficult until the magnesium deficiency has been corrected

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

[Hypokalemia - causes, symptoms, diagnosis, treatment, pathology]

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **56.8%**

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👍 👎 Discuss (1) Improve

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+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypokalemia - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 4 1

Report broken media

Score: **56.8%**

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A 60-year-old woman had a thyroid function test requested by her General Practitioner after reporting some symptoms of mild lethargy. This had unexpectedly demonstrated a suppressed Thyroid Stimulating Hormone level (0.25 microU / L) but normal free T4 level (14.1 pmol / L). She denied any heat intolerance, weight loss, diarrhoea, hair or skin changes, palpitations or eye symptoms.

The patient had a hysterectomy without oophorectomy at age 45 as a treatment for menorrhagia secondary to fibroids. She remembers reaching menarche at around the age of 13 or 14 years. There is no significant family history of coronary artery disease. The patient reported that her mother had suffered a fractured neck of femur at the age of 75 years following a fall. The patient was a retired school teacher with an active lifestyle. She had never smoked and drank very little alcohol.

Examination showed no evidence of a goitre, no fine tremor and no lid lag. External examination of the eyes was unremarkable. The cardiovascular and respiratory examination was unremarkable.

The GP requested some further basic investigations and then repeated blood tests 2 months after the original test. At this time, the patient reported her previous symptoms of lethargy had improved; with hindsight, she attributed this to grief due to the recent death of a close friend.

Ambulatory blood pressure monitoring: average blood pressure 125 / 75 mmHg

ECG: sinus rhythm at 75 bpm; normal axis; no abnormality of QRS, ST interval or T waves.

Haemoglobin	12.8 g / dL
White cell count	6.5 x 10^9/l
Platelets	206 x 10^9/l

Urea	6.2 mmol / L
Creatinine	95 micromol / L
Sodium	137 mmol / L
Potassium	4.0 mmol / L
C-reactive protein	< 1
Parathyroid hormone	3.7 pmol / L (reference 1.2-5.8)
Thyroid-stimulating hormone	0.21 microU / L (reference 0.4-5.0)
T4 free serum	13.8 pmol / L (reference 8.5-15.2)
T3 free serum	5.6 pmol / L (reference 3.5-6.5)
HbA1C	5.6 % (reference 4-6)
Total cholesterol	4.0 mmol / L
LDL cholesterol	1.8 mmol / L
HDL cholesterol	1.9 mmol / L

What is the most appropriate management of the deranged thyroid function tests?

- ○ DEXA scan ✕
- ○ Thyroid ultrasound ✕
- ○ Start treatment with simvastatin ✕
- ○ Radioiodine therapy ✕
- ○ Treat with propylthiouracil ✕

Submit answer

Reference ranges ⌄

Score: **56.8%**

1	✓
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75	-

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Question 75 of 342 ✗

A 60-year-old woman had a thyroid function test requested by her General Practitioner after reporting some symptoms of mild lethargy. This had unexpectedly demonstrated a suppressed Thyroid Stimulating Hormone level (0.25 microU / L) but normal free T4 level (14.1 pmol / L). She denied any heat intolerance, weight loss, diarrhoea, hair or skin changes, palpitations or eye symptoms.

The patient had a hysterectomy without oophorectomy at age 45 as a treatment for menorrhagia secondary to fibroids. She remembers reaching menarche at around the age of 13 or 14 years. There is no significant family history of coronary artery disease. The patient reported that her mother had suffered a fractured neck of femur at the age of 75 years following a fall. The patient was a retired school teacher with an active lifestyle. She had never smoked and drank very little alcohol.

Examination showed no evidence of a goitre, no fine tremor and no lid lag. External examination of the eyes was unremarkable. The cardiovascular and respiratory examination was unremarkable.

The GP requested some further basic investigations and then repeated blood tests 2 months after the original test. At this time, the patient reported her previous symptoms of lethargy had improved; with hindsight, she attributed this to grief due to the recent death of a close friend.

Ambulatory blood pressure monitoring: average blood pressure 125 / 75 mmHg

ECG: sinus rhythm at 75 bpm; normal axis; no abnormality of QRS, ST interval or T waves.

Haemoglobin	12.8 g / dL
White cell count	6.5 x 10^9/l
Platelets	206 x 10^9/l

Urea	6.2 mmol / L
Creatinine	95 micromol / L
Sodium	137 mmol / L
Potassium	4.0 mmol / L
C-reactive protein	< 1
Parathyroid hormone	3.7 pmol / L (reference 1.2-5.8)
Thyroid-stimulating hormone	0.21 microU / L (reference 0.4-5.0)
T4 free serum	13.8 pmol / L (reference 8.5-15.2)
T3 free serum	5.6 pmol / L (reference 3.5-6.5)
HbA1C	5.6 % (reference 4-6)
Total cholesterol	4.0 mmol / L
LDL cholesterol	1.8 mmol / L
HDL cholesterol	1.9 mmol / L

What is the most appropriate management of the deranged thyroid function tests?

🟩	DEXA scan	32%
🟥	Thyroid ultrasound	43%
	Start treatment with simvastatin	8%
	Radioiodine therapy	4%
	Treat with propylthiouracil	14%

The patient has subclinical hyperthyroidism with persistently suppressed TSH levels but normal serum thyroid hormone levels and with no clinical evidence of thyrotoxicosis. This usually occurs in the setting of thyroid overactivity due to Graves' disease or autonomously functioning thyroid nodules sufficient to suppress pituitary TSH secretion but insufficient to cause an elevation of circulating hormones. Progression to overt hyperthyroidism occurs in 1-3 % of elderly patients per year.

The main risk of subclinical hyperthyroidism is the increased risk of atrial fibrillation and hip fractures. The American Association of Clinical Endocrinologists recommends that treatment is considered in patients with a persistently low TSH level if they are older than 65 years or are at risk of osteoporosis or heart disease.

This patient has a low level of cardiac risk factors with a low-risk lipid profile. Assessment of her osteoporosis risk is complicated by her hysterectomy preventing knowledge of her age at menopause. Therefore, a DEXA scan is appropriate next line management to quantify her osteoporosis risk and inform the decision as to whether or not to treat the sub-clinical hyperthyroidism.

Thyroid ultrasound would not influence the decision to treat at this stage and so is not required.

Weetman A. Investigating low thyroid stimulating hormone (TSH) level. BMJ 2013;347:f6842.

👍 👎 Discuss (9) Improve

Next question

Subclinical hyperthyroidism ★

Subclinical hyperthyroidism is an entity which is gaining increasing recognition. It is defined as:

- normal serum free thyroxine and triiodothyronine levels
- with a thyroid stimulating hormone (TSH) below normal range (usually < 0.1 mu/l)

Causes

- multinodular goitre, particularly in elderly females
- excessive thyroxine may give a similar biochemical picture

The importance in recognising subclinical hyperthyroidism lies in the potential effect on the cardiovascular system (atrial fibrillation) and bone metabolism (osteoporosis). It may also impact on quality of life and increase the likelihood of dementia

Management

- TSH levels often revert to normal - therefore levels must be persistently low to warrant intervention
- a reasonable treatment option is a therapeutic trial of low-dose antithyroid agents for approximately 6 months in an effort to induce a remission

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Dr. Jamil Alrajhi

Score: **56%**

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75 ✗

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A 45-year-old woman presents to her GP with a 6-month history of fatigue, constipation, and generalised bone pain. She also reports increased thirst and urination. On examination, she appears well but has mild tenderness over her lower back. Blood tests are performed:

Calcium	2.85 mmol/L	(2.2 - 2.6)
Phosphate	0.7 mmol/L	(0.8 - 1.5)
PTH	9.5 pmol/L	(1.6 - 6.9)
Alkaline phosphatase	180 U/L	(30 - 130)
Creatinine	95 μmol/L	(45 - 90)

A DEXA scan shows osteopenia with a T-score of -2.1 at the lumbar spine. A renal ultrasound reveals a 5mm calculus in the right kidney.

What is the most appropriate management for this patient?

- ○ Oral calcium and vitamin D supplementation ×
- ○ Cinacalcet therapy ×
- ○ Parathyroidectomy ×
- ○ Bisphosphonate therapy ×
- ○ Annual monitoring with blood tests and DEXA scans ×

Submit answer

Reference ranges

Score: **56%**

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A 21-year-old woman presents to the Emergency department following a collapse at the local supermarket. She tells you that she always feels weak and washed out, and hardly ever has any energy. She takes no regular medication and virtually never sees the doctor. On examination her blood pressure is 100/70 mmHg, pulse is 80 beats per minute and regular. She is slim with a body mass index of 21 kg/m², no abnormal physical signs are noted.

Investigations

Na^+	140 mmol/l
K^+	3.1 mmol/l
$HCO3^-$	32 mmol/l
Urea	5.9 mmol/l
Creatinine	85 μmol/l

Which of the following is the most likely diagnosis?

- ○ Conn's syndrome ×
- ○ Cushing's syndrome ×
- ○ Gitelman's syndrome ×
- ○ Liddle's syndrome ×
- ○ Renal tubular acidosis type 1 ×

Submit answer

Reference ranges

Score: **55.3%**

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Investigations

Na^+	140 mmol/l
K^+	3.1 mmol/l
$HCO3^-$	32 mmol/l
Urea	5.9 mmol/l
Creatinine	85 µmol/l

Which of the following is the most likely diagnosis?

Conn's syndrome	6%
Cushing's syndrome	1%
Gitelman's syndrome	74%
Liddle's syndrome	10%
Renal tubular acidosis type 1	9%

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

Important for me Less important

This patient has hypokalaemic metabolic alkalosis with normal / low blood pressure. This fits best with a diagnosis of Gitelman's syndrome, Bartter's syndrome, or diuretic abuse. Bartter's syndrome often presents in infancy as failure to thrive, and isn't listed as a distractor. Diuretic abuse presents with a similar clinical picture although it too isn't listed here as an option. Gitelman's is caused by a mutation in the gene coding for the thiazide-sensitive sodium-chloride co-transporter, and results in a clinical picture similar to that expected with thiazide diuretic use.

Conn's syndrome is associated with hypertension, as is Liddle's syndrome, effectively ruling them out as diagnoses here. Renal tubular acidosis type 1 is associated with hypokalaemia and metabolic acidosis, not with a bicarbonate of 32.

👍 👎 Discuss (3) Improve

Next question

Gitelman's syndrome

Gitelman's syndrome is due to a defect in the thiazide-sensitive Na$^+$ Cl- transporter in the distal convoluted tubule.

Features
- normotension
- hypokalaemia
- hypocalciuria
- hypomagnesaemia
- metabolic alkalosis

Diagnosis
- genetic testing for mutations in the SLC12A3 gene

Management
- oral potassium and magnesium supplementation
- potassium-sparing diuretics may be used if still hypokalaemic/symptomatic

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.8%**

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77	✓

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Question 78 of 342

A 62-year-old woman presents to hospital. She has been agitated over the last few days and her daughter has become concerned about this. She complains of nausea and has vomited once. She also has a cough which has been productive with a green sputum, and she has had some chest pain when she coughs. She also is confused and agitated. On examination, she has a heart rate of 150bpm, blood pressure of 103/65mmHg, temperature 40.1° and oxygen saturation of 94%. She has a past medical history of myocardial infarction three years ago needing cardiac stenting, polycystic ovaries and hypertension. She has also diagnosed with hyperthyroidism four months ago but she admits poor compliance with treatment. She is supposed to take aspirin, atorvastatin, amlodipine and carbimazole. Her blood tests are pending. What feature of her presentation is most suggestive of thyroid storm?

○ Productive cough ×

○ History of aspirin use ×

○ Chest pain ×

○ Confusion ×

○ Hyperpyrexia ×

Submit answer

Reference ranges

Score: **55.8%**

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78	-

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Productive cough	1%
History of aspirin use	9%
Chest pain	1%
Confusion	13%
[✓ Hyperpyrexia]	76%

In thyroid storm, hyperpyrexia, tachycardia, cardiac failure and agitation are the key diagnostic elements

Important for me Less important

This patient has features of thyroid storm. The most important features are high fever and tachycardia, with temperatures becoming very high, even above 40ºC, and heart rate can be as high as 180bpm. Thyroid storm can be triggered by infection, and therefore the productive cough is relevant but not a distinguishing feature. The use of aspirin can worsen thyrotoxicosis as well by displacing T4 from thyroid binding globulin but is not a main feature. Agitation and confusion are both features but are less discriminating than hyperpyrexia.

Discuss (2) Improve

Next question

Thyroid storm

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:
- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine

- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **56.4%**

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Question 79 of 342

A 28-year-old woman presents to the clinic with amenorrhea. She had a regular menstrual cycle up until 4 months ago. On examination, you note a male pattern of her growth.

Blood results are as follows:

Testosterone	2.2 nmol/L	(< 1.8)
Oestradiol	34 pg/ml	(25-75)
FSH	12 IU/L	(1.8 - 22.5)
LH	84 IU/L	(1.2-103)

What is the most likely diagnosis?

- ○ Androgen-secreting tumour ×
- ○ Kallmann's syndrome ×
- ○ Partial androgen insensitivity syndrome (PAIS) ×
- ○ Polycystic ovary syndrome (PCOS) ×
- ○ Turner's syndrome ×

Submit answer

Reference ranges

Score: **56.4%**

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Question 79 of 342 ✓

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Blood results are as follows:

Testosterone	2.2 nmol/L	(< 1.8)
Oestradiol	34 pg/ml	(25-75)
FSH	12 IU/L	(1.8 - 22.5)
LH	84 IU/L	(1.2-103)

What is the most likely diagnosis?

Androgen-secreting tumour	21%
Kallmann's syndrome	3%
Partial androgen insensitivity syndrome (PAIS)	21%
Polycystic ovary syndrome (PCOS)	54%
Turner's syndrome	1%

Stereotypical PCOS results:

- raised LH:FSH ratio
- testosterone may be normal or mildly elevated
- SHBG is normal to low

Important for me Less important

Polycystic ovary syndrome (PCOS) is correct. The patient has several clinical features of PCOS including secondary amenorrhea and hirsutism. The mildly elevated testosterone and raised LH:FSH ratio further support the diagnosis.

Androgen-secreting tumour is incorrect. An androgen-secreting tumour would result in a more profound increase in testosterone levels resulting in virilisation.

Kallmann's syndrome is incorrect. Kallmann's syndrome is characterised by primary amenorrhea secondary to hypogonadotropic hypogonadism. However, in this case, the biochemical profile is not in keeping with Kallmann's syndrome - which would present with low oestradiol and inappropriately low/normal LH, and FSH levels.

Partial androgen insensitivity syndrome (PAIS) is incorrect. PAIS results in a spectrum of disorders that ranges from a female phenotype with clitoromegaly and/or minimal posterior labial fusion (Lub syndrome) to genital ambiguity to an unequivocally male phenotype with a minor defect such as isolated hypospadias or azoospermia. There are no clinical features in this case to support this diagnosis.

Turner's syndrome is incorrect. Turner's syndrome is a cause of primary ovarian failure. It would present with primary amenorrhoea, rather than secondary amenorrhea seen in this case. Furthermore, the biochemical profile, in this case, is not in keeping with Turner's syndrome - which would present with low sex hormone levels and markedly raised LH and FSH levels.

Discuss (6) Improve

Next question

Polycystic ovarian syndrome: features and investigation

Polycystic ovary syndrome (PCOS) is a complex condition of ovarian dysfunction thought to affect between 5-20% of women of reproductive age. The aetiology of PCOS is not fully understood. Both hyperinsulinaemia and high levels of luteinizing hormone are seen in

PCOS and there appears to be some overlap with the metabolic syndrome.

Features

- subfertility and infertility
- menstrual disturbances: oligomenorrhoea and amenorrhoea
- hirsutism, acne (due to hyperandrogenism)
- obesity
- acanthosis nigricans (due to insulin resistance)

Investigations

- pelvic ultrasound: multiple cysts on the ovaries
- FSH, LH, prolactin, TSH, testosterone, sex hormone-binding globulin (SHBG) are useful investigations
  - raised LH:FSH ratio is a 'classical' feature but is no longer thought to be useful in diagnosis
  - prolactin may be normal or mildly elevated
  - testosterone may be normal or mildly elevated - however, if markedly raised consider other causes
  - SHBG is normal to low in women with PCOS
- check for impaired glucose tolerance

Diagnostic criteria

- a formal diagnosis should only be made after performing investigations to exclude other conditions
- the Rotterdam criteria state that a diagnosis of PCOS can be made if 2 of the following 3 are present:
  - infrequent or no ovulation (usually manifested as infrequent or no menstruation)
  - clinical and/or biochemical signs of hyperandrogenism (such as hirsutism, acne, or elevated levels of total or free testosterone)
  - polycystic ovaries on ultrasound scan (defined as the presence of ≥ 12 follicles (measuring 2-9 mm in diameter) in one or both ovaries and/or increased ovarian volume $> 10\ \text{cm}^3$)

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 8 👎 8

Polycystic ovarian syndrome review

Royal College of Obstetricians and Gynaecologists 👍 4 👎 3

2014 Long-term Consequences of Polycystic Ovary Syndrome

Clinical Knowledge Summaries 👍 4 👎 3

Polycystic ovarian syndrome

Suggest link

Report broken link

Score: **57%**

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A 76-year-old man with advanced prostate cancer and bone metastases is admitted to hospital, complaining of generalised abdominal pain and presenting with some confusion. A number of blood tests are performed, including the following:

Calcium	3.2 mmol/L	(2.1-2.6)

Aggressive intravenous fluid rehydration is administered (4-6 litres per day), which is unsuccessful. Pamidronic acid is also tried, which is unsuccessful, as a repeat blood test shows a similar result:

Calcium	3.18 mmol/L	(2.1-2.6)

What is most reasonable to try next?

- ○ Calcitonin
- ○ Furosemide
- ○ Ibandronic acid
- ○ Increase intravenous fluids
- ○ Prednisolone

Submit answer

Reference ranges

Score: **57%**

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Question 80 of 342 ✗

A 76-year-old man with advanced prostate cancer and bone metastases is admitted to hospital, complaining of generalised abdominal pain and presenting with some confusion. A number of blood tests are performed, including the following:

Calcium	3.2 mmol/L	(2.1-2.6)

Aggressive intravenous fluid rehydration is administered (4-6 litres per day), which is unsuccessful. Pamidronic acid is also tried, which is unsuccessful, as a repeat blood test shows a similar result:

Calcium	3.18 mmol/L	(2.1-2.6)

What is most reasonable to try next?

🟩	Calcitonin	75%
	Furosemide	8%
🟥	Ibandronic acid	10%
	Increase intravenous fluids	2%
	Prednisolone	6%

Refractory hypercalcaemia of malignancy may be treated with subcutaneous calcitonin if therapy with fluids and pamidronate fails

Important for me Less important

The case above is an example of refractory hypercalcaemia on a background of malignancy. As intravenous fluid treatment is unsuccessful, bisphosphonates are tried next. Given that these are also unsuccessful, the most suitable step to try next, given the patient's history, is **calcitonin**. This is administered subcutaneously.

Furosemide is a loop diuretic. These are sometimes used in hypercalcaemia, particularly those who cannot tolerate aggressive fluid rehydration. However, in this patient, calcitonin is a more suitable choice.

Ibandronic acid is another example of a bisphosphonate. Given that pamidronic acid has already been tried, and was unsuccessful, calcitonin would be a more reasonable step at this stage, rather than trying another bisphosphonate.

Increasing fluids beyond the current 4-6 litres per 24 hour period would not be appropriate. The patient is already taking a large volume of fluid and further increase may lead to overload.

Prednisolone is a steroid and may be used if the patient had a background of sarcoidosis or other granulomatous disease, rather than malignancy.

👍 👎 Discuss (5) Improve

Next question

Hypercalcaemia: management ★

The initial management of hypercalcaemia is rehydration with normal saline, typically 3-4 litres/day. Following rehydration bisphosphonates may be used. They typically take 2-3 days to work with maximal effect being seen at 7 days

Other options include:
- calcitonin - quicker effect than bisphosphonates
- steroids in sarcoidosis

Loop diuretics such as furosemide are sometimes used in hypercalcaemia, particularly in patients who cannot tolerate aggressive fluid rehydration. However, they should be used with caution as they may worsen electrolyte derangement and volume depletion.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 4 👎 7

Hypercalcaemia â€“ presentation and management

Suggest link Report broken link

Media

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 0 👎 1

Report broken media

Score: **56.3%**

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← Question 81 of 342 →

A 17-year-old woman presents to the clinic with amenorrhea. She advises you that she has never had a menstrual cycle. On examination, you note little axillary and pubic hair. There are also bilateral groin swellings. Her vagina and labia have a normal appearance.

Blood results are as follows:

Testosterone	11.2 nmol/L	(< 1.8)
FSH	16 IU/L	(1.8 - 22.5)
LH	120 IU/L	(1.2-103)

What is the most likely diagnosis?

- ○ Complete androgen insensitivity syndrome (CAIS) ×
- ○ Kallmann's syndrome ×
- ○ Mullerian agenesis ×
- ○ Partial androgen insensitivity syndrome (PAIS) ×
- ○ Turner's syndrome ×

Submit answer

Reference ranges

Score: **56.3%**

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A 17-year-old woman presents to the clinic with amenorrhea. She advises you that she has never had a menstrual cycle. On examination, you note little axillary and pubic hair. There are also bilateral groin swellings. Her vagina and labia have a normal appearance.

Blood results are as follows:

Testosterone	11.2 nmol/L	(< 1.8)
FSH	16 IU/L	(1.8 - 22.5)
LH	120 IU/L	(1.2-103)

What is the most likely diagnosis?

🟩	Complete androgen insensitivity syndrome (CAIS)	62%
🟥	Kallmann's syndrome	4%
	Mulerian agenesis	4%
	Partial androgen insensitivity syndrome (PAIS)	26%
	Turner's syndrome	4%

Primary amenorrhoea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome

Important for me Less important

Complete androgen insensitivity syndrome (CAIS) is correct. The clinical features of primary amenorrhea, lack of secondary sexual characteristics, and groin swellings (suggestive of undescended testes) are suggestive of androgen insensitivity syndrome. The hormonal profile findings of very high testosterone, elevated LH, and a relatively normal

FSH are classical findings of partial androgen insensitivity syndrome. The presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome.

Kallmann's syndrome is incorrect. Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. Cryptorchidism can also occur. However, the biochemical profile, in this case, is not in keeping with Kallmann's syndrome - which would present with low sex hormone levels, and inappropriately low/normal LH, and FSH levels.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Both Mullerian agenesis and androgen insensitivity syndrome can present with primary amenorrhea, shortened vagina, and absent cervix. However, in patients with androgen insensitivity, the gonads are testes, which produce normal androgens. The presence of bilateral groin swellings (indicative of undescended testes) makes Mullerian agenesis a less likely diagnosis.

Partial androgen insensitivity syndrome (PAIS) is incorrect. The clinical and biochemical features are suggestive of androgen insensitivity syndrome. However the presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome. PAIS results in a spectrum of disorders that ranges from a female phenotype with clitoromegaly and/or minimal posterior labial fusion (Lub syndrome) to genital ambiguity to an unequivocally male phenotype with a minor defect such as isolated hypospadias or azoospermia.

Turner's syndrome is incorrect. Turner's syndrome can cause primary ovarian failure due to gonadal dysgenesis. However, the biochemical profile, in this case, is not in keeping with Turner's syndrome - which would present with low sex hormone levels, and markedly raised LH and FSH levels.

👍 | 👎 | 💬 Discuss (2) | Improve

Next question

Androgen insensitivity syndrome

Androgen insensitivity syndrome is an X-linked recessive condition due to end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Complete androgen insensitivity syndrome is the new term for testicular feminisation syndrome

Features

- 'primary amenorrhoea'
- little or no axillary and pubic hair
- undescended testes causing groin swellings
- breast development may occur as a result of the conversion of testosterone to oestradiol

Diagnosis

- buccal smear or chromosomal analysis to reveal 46XY genotype
- after puberty, testosterone concentrations are in the high-normal to slightly elevated reference range for postpubertal boys

Management

- counselling - raise the child as female
- bilateral orchidectomy (increased risk of testicular cancer due to undescended testes)
- oestrogen therapy

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.6%**

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Question 82 of 342

A 19-year-old woman presents for review. Her past medical history includes 11-beta-hydroxylase deficiency and hypertension, which is managed with ramipril and indapamide. The 11-beta-hydroxylase deficiency was identified at birth upon identifying clitoromegaly.

Which of the following is likely to be raised most markedly?

- ○ 17-OH pregnenolone ✕
- ○ Oestradiol ✕
- ○ 11-deoxycortisol ✕
- ○ 17-OH progesterone ✕
- ○ Oestrone ✕

Submit answer

Reference ranges

Score: **55.6%**

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← Question 82 of 342 ✗ →

A 19-year-old woman presents for review. Her past medical history includes 11-beta-hydroxylase deficiency and hypertension, which is managed with ramipril and indapamide. The 11-beta-hydroxylase deficiency was identified at birth upon identifying clitoromegaly.

Which of the following is likely to be raised most markedly?

	17-OH pregnenolone	12%
	Oestradiol	7%
🟩	11-deoxycortisol	32%
🟥	17-OH progesterone	49%
	Oestrone	1%

11 beta-hydroxylase is responsible for the conversion of 11-deoxycorticosterone and 11-deoxycortisol to corticosterone and cortisol. In patients with 11-beta-hydroxylase deficiency, this conversion does not occur in sufficient amounts and levels of these steroids accumulate in the patient. Therefore, although 17-OH hormones may also be raised, the 11-deoxycortisol is more significantly raised than the others.

Discuss (7) | Improve

Next question

Congenital adrenal hyperplasia ★

Congenital adrenal hyperplasia (CAH) refers to a group of autosomal recessive disorders that impair adrenal steroid biosynthesis. The deficiency in cortisol production leads to compensatory overproduction of adrenocorticotropic hormone (ACTH) by the anterior pituitary. Elevated ACTH levels increase the production of adrenal androgens, which can result in the virilization of female infants and affect genital development.

Cause

- 21-hydroxylase deficiency (90%)
  - impairs the conversion of 17-hydroxyprogesterone to 11-deoxycortisol, leading to cortisol deficiency and excess androgen production
- 11-beta hydroxylase deficihttps://www.passmedicine.com/database/dbnotes.php#ency (5%)
  - results in hypertension due to excess deoxycorticosterone
- 17-hydroxylase deficiency (very rare)
  - leads to mineralocorticoid excess with low androgen and estrogen levels

Clinical features

- the symptoms and signs of CAH vary depending on the specific enzyme deficiency and the severity of the disorder. Common clinical presentations include:
- virilization:
  - female infants may present with ambiguous genitalia due to excessive androgen exposure in utero.
  - male infants appear normal at birth, which can delay diagnosis.
- salt-wasting crisis:
  - this severe form occurs in about 75% of cases with 21-hydroxylase deficiency
  - it is characterized by dehydration, hypotension, and electrolyte imbalances, and can be life-threatening if not treated promptly.
- precocious puberty: Excess androgens can lead to early development of secondary sexual characteristics in both males and females.
- infertility: Adults with untreated CAH may experience fertility issues due to hormonal imbalances.

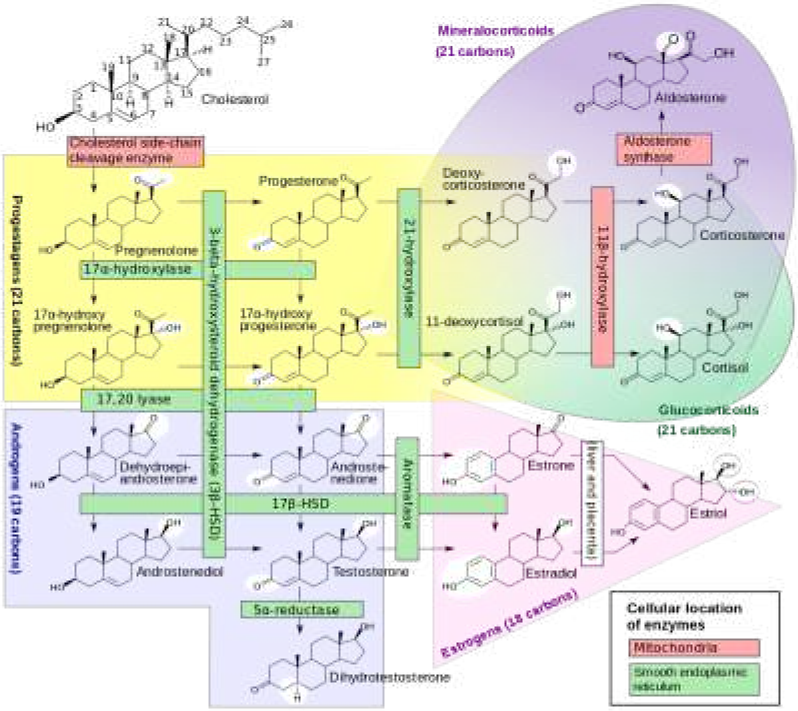
- height and growth abnormalities: Children with CAH often experience accelerated growth rates initially but may have a shorter adult stature due to early epiphyseal closure.

Diagnosis

- many countries screen newborns for CAH, looking for elevated levels of serum concentration of 17-hydroxyprogesterone (17OHP). This is however not yet done in the UK
- ACTH stimulation testing is used to confirm the diagnosis
    - evaluates the adrenal gland's response to ACTH, with abnormal increases in 17OHP indicating CAH

Management

- primarily involves glucocorticoid replacement to reduce ACTH levels and minimize adrenal androgen production
- in cases of mineralocorticoid deficiency fludrocortisone is also prescribed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Cholesterol side-chain cleavage enzyme
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Estrone
liver and placenta
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Testosterone
Estradiol
5α-reductase
Dihydrotestosterone
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Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

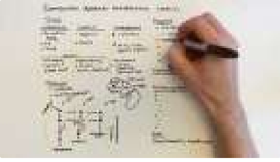
Next question

Textbooks

High-yield textbook

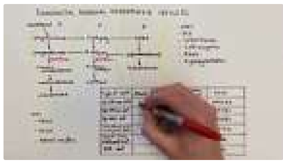
Extended textbook

Media



Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 👍 18 👎 2



Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 👍 13 👎 2

Report broken media

Score: **54.9%**

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82	✗

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A 47-year-old female presents with recurrent palpitations. She also complains of associated sweating, blurring of vision and generalized weakness. She denies any chest pain, shortness of breath or loss of consciousness. She had several similar episodes over last few months. The episodes usually occur in the morning or just before a meal. She has noticed that her symptoms improve after she eats something. She has gained weight of about 9 kg over last few months. She has a past history of an anxiety disorder but is not currently taking any regular medications. She does not smoke and only occasionally drinks alcohol. Her blood glucose at present is 4.9 mmol. You suspect insulinoma based on the history.

What is the next step in the evaluation of this patient?

- ○ Check insulin level ×
- ○ Check pro-insulin level ×
- ○ CT abdomen ×
- ○ Screening for sulphonylurea drugs ×
- ○ Check blood glucose during or after the episode to confirm hypoglycemia ×

Submit answer

Reference ranges

Score: **54.9%**

1	✓
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Question 83 of 342 ✓

A 47-year-old female presents with recurrent palpitations. She also complains of associated sweating, blurring of vision and generalized weakness. She denies any chest pain, shortness of breath or loss of consciousness. She had several similar episodes over last few months. The episodes usually occur in the morning or just before a meal. She has noticed that her symptoms improve after she eats something. She has gained weight of about 9 kg over last few months. She has a past history of an anxiety disorder but is not currently taking any regular medications. She does not smoke and only occasionally drinks alcohol. Her blood glucose at present is 4.9 mmol. You suspect insulinoma based on the history.

What is the next step in the evaluation of this patient?

Check insulin level	12%
Check pro-insulin level	39%
CT abdomen	10%
Screening for sulphonylurea drugs	3%
Check blood glucose during or after the episode to confirm hypoglycemia	36%

Completion of Whipple's triad is required before further investigations for insulinoma

Important for me Less important

European Neuroendocrine Tumor Society (ENETS) guidelines published in 2016 outline the diagnostic criteria for insulinomas. As symptoms of hypoglycemia can be non-specific and attributable to several different conditions, it is important to confirm hypoglycemia by checking blood glucose. Therefore, completion of Whipple's triad is required before embarking on further investigations. Whipple's triad is defined as the presence of hypoglycemic symptoms, accompanying low blood glucose, and resolution of symptoms after correcting the blood glucose levels.

Therefore, the best answer here is 5 i.e confirming the hypoglycemia will be the next step in the evaluation of this patient.

All the other options are relevant to the diagnosis but not the best next step here.

👍 👎 Discuss (8) Improve

Next question

Insulinoma ★

An insulinoma is a neuroendocrine tumour deriving mainly from pancreatic Islets of Langerhans cells

Basics
- most common pancreatic endocrine tumour
- 10% malignant, 10% multiple
- of patients with multiple tumours, 50% have MEN-1

Features
- of hypoglycaemia: typically early in morning or just before meal, e.g. diplopia, weakness etc
- rapid weight gain may be seen
- high insulin, raised proinsulin:insulin ratio
- high C-peptide

Diagnosis
- supervised, prolonged fasting (up to 72 hours)
- CT pancreas

Management

- surgery
- diazoxide and somatostatin if patients are not candidates for surgery

+ Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media

PANCREATIC
NEUROENDOCRINE
NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **55.4%**

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Question 84 of 342

A 22-year-old woman presents with persistent nausea and vomiting. She is 16 weeks gestation. She has no significant past medical history and takes no regular medicines. Observations are within normal limits.

Blood results are as follows:

Hb	115 g/L	Male: (135-180) Female: (115 - 160)
Platelets	192 * 10^9/L	(150 - 400)
WBC	10.2 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	3.5 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	56 μmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What treatment is indicated?

- ○ Cyclizine ×
- ○ Domperidone ×
- ○ Metoclopramide ×
- ○ Ondansetron ×
- ○ Pyridoxine ×

Submit answer

Reference ranges ∨

Score: **55.4%**

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79	✓
80	✗
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83	✓
84	-

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← Question 84 of 342 ✓ →

A 22-year-old woman presents with persistent nausea and vomiting. She is 16 weeks gestation. She has no significant past medical history and takes no regular medicines. Observations are within normal limits.

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K^+	3.5 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	56 μmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What treatment is indicated?

Cyclizine	63%
Domperidone	5%
Metoclopramide	9%
Ondansetron	12%
Pyridoxine	11%

Antihistamines are first-line in the management of nausea & vomiting in pregnancy/hyperemesis gravidarum

Important for me Less important

Cyclizine is correct. The royal college of Obstetrics and Gynaecology (RCOG) guidelines recommend antihistamines as the first-line agents for nausea and vomiting in pregnancy.

Domperidone is incorrect. This drug is a second-line agent. The risk of extrapyramidal side effects is less compared to metoclopramide.

Metoclopramide is incorrect. This drug is a second-line agent. Metoclopramide can cause extrapyramidal side effects, particularly in young females (<25 years old).

Ondansetron is incorrect. This drug is a second-line agent. The use of ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate.

Pyridoxine is incorrect. Pyridoxine can be used in conjunction with doxylamine, however, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines.

👍 👎 Discuss (1) Improve

Next question

Hyperemesis gravidarum ★

Whilst the majority of women experience nausea (previously termed 'morning sickness') during the early stages of pregnancy it can become problematic in a minority of cases. The Royal College of Obstetricians and Gynaecologists (RCOG) now use the term 'nausea and vomiting of pregnancy' (NVP) to describe troublesome symptoms, with hyperemesis gravidarum being the extreme form of this condition.

It occurs in around 1% of pregnancies and is thought to be related to raised beta hCG levels. Hyperemesis gravidarum is most common between 8 and 12 weeks but may persist up to 20 weeks.

Risk factors

- increased levels of beta-hCG

    - multiple pregnancies
    - trophoblastic disease
- nulliparity
- obesity
- family or personal history of NVP

Smoking is associated with a decreased incidence of hyperemesis.

Referral criteria for nausea and vomiting in pregnancy

NICE recommend considering admission in the following situations:
- Continued nausea and vomiting and is unable to keep down liquids or oral antiemetics
- Continued nausea and vomiting with ketonuria and/or weight loss (greater than 5% of body weight), despite treatment with oral antiemetics
- A confirmed or suspected comorbidity (for example she is unable to tolerate oral antibiotics for a urinary tract infection)

They also recommend having a lower threshold for admission to hospital if the woman has a co-existing condition (for example diabetes) that may be adversely affected by nausea and vomiting.

Hyperemesis gravidarum

The Royal College of Obstetricians and Gynaecologists (RCOG) recommend that the following triad is present before diagnosis of hyperemesis gravidarum: RCOG
- 5% pre-pregnancy weight loss
- dehydration
- electrolyte imbalance

Validated scoring systems such as the Pregnancy-Unique Quantification of Emesis (PUQE) score can be used to classify the severity of NVP.

Management

- simple measures
    - rest and avoid triggers e.g. odours
    - bland, plain food, particularly in the morning
    - ginger
    - P6 (wrist) acupressure
- first-line medications
    - antihistamines: oral cyclizine or promethazine
    - phenothiazines: oral prochlorperazine or chlorpromazine
    - combination drug doxylamine/pyridoxine: pyridoxine (vitamin B6) monotherapy is actually used commonly outside of the UK as a first-line treatment for NVP. However, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines
- second-line medications
    - oral ondansetron: ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate. The Medicines and Healthcare products Regulatory Agency (MHRA) advise that if ondansetron is used then these risks should be discussed with the pregnant woman
    - oral metoclopramide or domperidone: metoclopramide may cause extrapyramidal side effects. It should therefore not be used for more than 5 days
- admission may be needed for IV hydration
    - see criteria above for referral/admission
    - normal saline with added potassium is used to rehydrate

Women with hyperemesis gravidarum may develop dehydration, weight loss and electrolyte imbalances. Other complications include:

- acute kidney injury
- Wernicke's encephalopathy
- oesophagitis, Mallory-Weiss tear
- venous thromboembolism
- fetal outcome
    - studies generally show little evidence of adverse outcomes for birth weight/other markers for mild-moderate symptoms
    - severe NVP resulting in multiple admissions and failure to 'catch-up' weight gain may be linked to a small increase in preterm birth and low birth weight

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Obstetricians and Gynaecologists 👍 11 👎 13

2016 The Management of Nausea and Vomiting of Pregnancy and Hyperemesis Gravidarum

Clinical Knowledge Summaries 👍 8 👎 4

CKS - NICE vomiting in pregnancy guidance summary

Suggest link

Report broken link

Score: **56%**

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← Question 85 of 342 →

A 43-year-old man is referred by his GP with a 4 week history of a lump appearing on the right side of his neck. The lump is roughly 7 mm and is located on the right side of the thyroid gland, in the anterior triangle. It does not move when the patient sticks out his tongue, but it does move on swallowing. There is no history of weight loss of night sweats.

Blood tests are performed and reveal:

Hb	12.9 g/l
Platelets	210 * 10^9/l
WBC	6.0 * 10^9/l
Na^+	141 mmol/l
K^+	3.9 mmol/l
Urea	4.1 mmol/l
Creatinine	33 µmol/l

What is the most appropriate first-line investigation?

- ○ Radioisotope scan of thyroid ×
- ○ Magnetic resonance scan of head and neck ×
- ○ Excision biopsy ×
- ○ Fine needle aspiration biopsy ×
- ○ Ultrasound scan of thyroid ×

Submit answer

Reference ranges

Score: **56%**

1	✓
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85	-

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Back to top

A 43-year-old man is referred by his GP with a 4 week history of a lump appearing on the right side of his neck. The lump is roughly 7 mm and is located on the right side of the thyroid gland, in the anterior triangle. It does not move when the patient sticks out his tongue, but it does move on swallowing. There is no history of weight loss of night sweats.

Blood tests are performed and reveal:

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Platelets	210 * 10^9/l
WBC	6.0 * 10^9/l
Na^+	141 mmol/l
K^+	3.9 mmol/l
Urea	4.1 mmol/l
Creatinine	33 µmol/l

What is the most appropriate first-line investigation?

Radioisotope scan of thyroid	8%
Magnetic resonance scan of head and neck	2%
Excision biopsy	1%
Fine needle aspiration biopsy	8%
[✓ Ultrasound scan of thyroid]	82%

High-resolution ultrasound scanning is an ideal first-line initial imaging investigation for most neck lumps. Because most lesions in the neck are site-specific, once a lesion has been located, specific ultrasound features can be used to establish the diagnosis.

👍 👎 Discuss (5) Improve

Next question

Thyrotoxicosis: causes and investigation

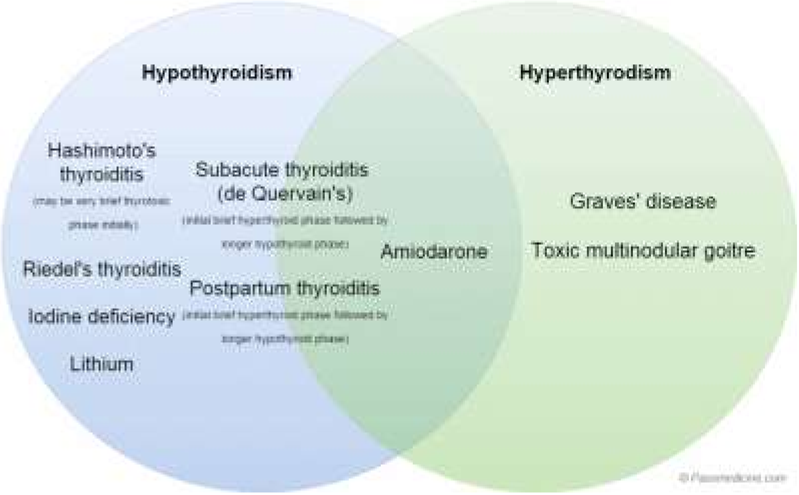
Graves' disease accounts for around 50-60% of cases of thyrotoxicosis.

Causes

- Graves' disease
- toxic nodular goitre
- acute phase of subacute (de Quervain's) thyroiditis
- acute phase of post-partum thyroiditis
- acute phase of Hashimoto's thyroiditis (later results in hypothyroidism)
- amiodarone therapy
- contrast
  - rare, more commonly occurs in elderly patients with pre-existing thyroid disease (e.g. multinodular goitre, Graves')
  - patients with existing thyrotoxicosis should not receive iodinated contrast medium
  - administration of iodinated contrast results in a large iodine load to the thyroid → hyperthyroidism developing over 2-12 weeks

Investigation

- TSH down, T4 and T3 up
- thyroid autoantibodies
- other investigations are not routinely done but includes isotope scanning


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Riedel's thyroiditis
Amiodarone
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
Graves' disease
Toxic multinodular goitre
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 2 4

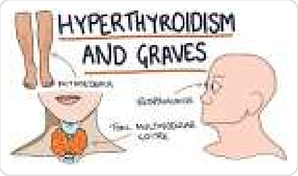
Hyperthyroidism guidelines

Royal College of Physicians 👍 7 👎 3

2017 Thyrotoxicosis â€“ investigation and management

Suggest link Report broken link

Media


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 2 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube 👍 0 👎 0

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Score: **56.5%**

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Question 86 of 342

A 42-year-old woman presents to her GP with a 6-month history of fatigue, reduced libido, and occasional headaches. She reports that her periods have become irregular over the past year. On examination, she has dry skin and appears pale. Visual field testing reveals a subtle bitemporal hemianopia. Blood tests are arranged.

TSH	0.8 mU/L	(0.5-5.0)
Free T4	8.2 pmol/L	(10.0-25.0)
Free T3	2.9 pmol/L	(3.5-6.5)
Prolactin	1250 mU/L	(< 500)
FSH	2.1 IU/L	(Follicular: 3.5-12.5)
LH	1.8 IU/L	(Follicular: 2.4-12.6)
Oestradiol	78 pmol/L	(Follicular: 98-571)
Cortisol (9am)	210 nmol/L	(170-620)

What is the most likely diagnosis?

- ○ Prolactinoma ×
- ○ Non-functioning pituitary adenoma ×
- ○ Primary hypothyroidism ×
- ○ Craniopharyngioma ×
- ○ Sheehan's syndrome ×

Submit answer

Reference ranges

Score: **56.5%**

1	✓
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A 42-year-old woman presents to her GP with a 6-month history of fatigue, reduced libido, and occasional headaches. She reports that her periods have become irregular over the past year. On examination, she has dry skin and appears pale. Visual field testing reveals a subtle bitemporal hemianopia. Blood tests are arranged.

TSH	0.8 mU/L	(0.5-5.0)
Free T4	8.2 pmol/L	(10.0-25.0)
Free T3	2.9 pmol/L	(3.5-6.5)
Prolactin	1250 mU/L	(< 500)
FSH	2.1 IU/L	(Follicular: 3.5-12.5)
LH	1.8 IU/L	(Follicular: 2.4-12.6)
Oestradiol	78 pmol/L	(Follicular: 98-571)
Cortisol (9am)	210 nmol/L	(170-620)

What is the most likely diagnosis?

Prolactinoma	26%
Non-functioning pituitary adenoma	72%
Primary hypothyroidism	1%
Craniopharyngioma	1%
Sheehan's syndrome	1%

The presence of an elevated prolactin level along with secondary hypothyroidism and hypogonadism is indicative of stalk compression is consistent with a non-functioning pituitary adenoma

Important for me Less important

Non-functioning pituitary adenoma is the most likely diagnosis in this case. The patient presents with symptoms and signs suggestive of multiple pituitary hormone deficiencies (secondary hypothyroidism, hypogonadism) along with features of mass effect (headaches and bitemporal hemianopia). The laboratory results show low-normal TSH with low free T4 and T3, indicating secondary hypothyroidism rather than primary hypothyroidism (where TSH would be elevated). The low gonadotropins (FSH and LH) with low oestradiol confirm hypogonadism of pituitary origin. The moderately elevated prolactin level (1250 mU/L) is significant but not markedly high enough to suggest a prolactinoma. This moderate hyperprolactinaemia is likely due to pituitary stalk compression, which disrupts the inhibitory dopamine signal from the hypothalamus to the pituitary, resulting in disinhibition of prolactin secretion - a phenomenon known as the 'stalk effect'. The morning cortisol is at the lower end of normal, suggesting possible early compromise of the ACTH-cortisol axis. Non-functioning pituitary adenomas typically present with symptoms of mass effect and hypopituitarism rather than hormone excess, which aligns with this clinical picture. The visual field defect (bitemporal hemianopia) is particularly suggestive of a pituitary mass compressing the optic chiasm. MRI imaging would be the next appropriate investigation to confirm the diagnosis.

Prolactinoma is less likely in this case. While the patient does have an elevated prolactin level, prolactinomas typically produce much higher prolactin levels, often >2000-3000 mU/L for macroadenomas. The moderate elevation seen here (1250 mU/L) is more consistent with stalk compression from a non-functioning adenoma. Additionally, while prolactinomas can cause hypogonadism, they are less likely to cause the broader pattern of hypopituitarism (including secondary hypothyroidism) seen in this patient.

Primary hypothyroidism does not explain this clinical picture. In primary hypothyroidism, TSH would be elevated rather than normal or low, and it wouldn't explain the hypogonadism, visual field defects, or elevated prolactin (although severe primary hypothyroidism can sometimes cause mild hyperprolactinaemia).

Craniopharyngioma could potentially cause similar symptoms through compression of the pituitary and optic chiasm. However, craniopharyngiomas are more common in children and young adults or in older adults (bimodal distribution), and often present with more pronounced symptoms of increased intracranial pressure, diabetes insipidus, and visual disturbances. While this remains in the differential diagnosis, a non-functioning pituitary adenoma is more likely in a 42-year-old woman with this presentation.

Sheehan's syndrome (postpartum pituitary necrosis) causes hypopituitarism following severe postpartum haemorrhage. There is no history of recent pregnancy or obstetric complications in this case, making Sheehan's syndrome unlikely. Additionally, Sheehan's syndrome would not explain the visual field defects, which suggest a space-occupying lesion.

👍 👎 💬 Discuss Improve

Next question

Pituitary adenoma ★

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:

- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due to excess prolactin)
- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
    - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
    - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).

    - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
    - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone) and neuromodulators like pasireotide
- transsphenoidal surgery
    - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
    - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
    - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
    - indicated for residual or recurrent tumours post-surgery

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info

👍 20 👎 11

Pituitary tumours review

Suggest link

Report broken link

Score: **57%**

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A 55-year-old woman is admitted following a fall whilst ice-skating, landing on her wrist and hitting her head on the ice. She felt slightly dizzy before the fall and has felt 'not quite right' for many months and was admitted to the hospital 1 month prior due to nephrolithiasis.

An x-ray confirms a scaphoid fracture. A CT head was unremarkable.

Hb	105 g/L	Male: (135-180) Female: (115 - 160)
Platelets	380 * 10^9/L	(150 - 400)
WBC	10.0 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	2.6 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	87 µmol/L	(55 - 120)
CRP	10 mg/L	(< 5)
Chloride	122 mmol/L	(101-110)
Bicarbonate	10 mmol/L	(22-28)

A venous blood gas is drawn:

pH	7.2	(7.35-7.45)
lactate	0.8 mmol/L	(0.0-2.0)
pC02	39 mmHg	(35 - 45)
p02	70 mmHg	(75-105)

Urine dip:

pH	6.5
Protein	trace

Blood	trace
Leucocytes	negative

From the following options, what would be the most likely cause of her biochemical abnormalities?

- ○ Carbonic anhydrase II deficiency ✕
- ○ Fanconi syndrome ✕
- ○ Hypoaldosteronism ✕
- ○ Sjogren's syndrome ✕
- ○ Topiramate use ✕

Submit answer

Reference ranges

Score: **57%**

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Question 87 of 342 ✗

A 55-year-old woman is admitted following a fall whilst ice-skating, landing on her wrist and hitting her head on the ice. She felt slightly dizzy before the fall and has felt 'not quite right' for many months and was admitted to the hospital 1 month prior due to nephrolithiasis.

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lactate	0.8 mmol/L	(0.0-2.0)
pC02	39 mmHg	(35 - 45)
p02	70 mmHg	(75-105)

Urine dip:

pH	6.5
Protein	trace

Blood	trace
Leucocytes	negative

From the following options, what would be the most likely cause of her biochemical abnormalities?

Carbonic anhydrase II deficiency	10%
Fanconi syndrome	27%
🟥 Hypoaldosteronism	4%
🟩 Sjogren's syndrome	55%
Topiramate use	3%

Type 1 (**distal**) renal tubular acidosis may be caused by Sjogren's syndrome

Important for me Less important

This patient has an unexplained metabolic acidosis with hypokalemia. If we calculate the anion gap we get a value of 10.6, indicating that this is a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include:
- Acetazolamide use
- Topiramate use
- Renal tubular acidosis type 1 and type 2
- Diarrhoea
- Ureterosigmoidostomy
- Post hypocapnic state

Sjogren's syndrome is correct. This is the only option that can cause type 1 renal tubular acidosis (RTA). Type 1 renal tubular acidosis is characterised by NAGMA, hypokalemia (due to renal potassium loss), high urinary pH (due to inability to acidify urine), and can also be a cause of renal stones. Type 1 RTA can be the first presentation of Sjogren's, so these patients should have autoimmune screening - anti-Ro-, anti-La, rheumatoid factor, and ANA.

Fanconi syndrome is incorrect. Although Fanconi syndrome can show NAGMA and hypokalemia, you would not expect to acidification of the urine as it is associated with type 2 RTA.

Hypoaldosteronism is incorrect. Hypoaldosteronism is associated with type 4 RTA and you would see hyperkalemia as aldosterone usually increases potassium excretion via the cortical collecting ducts.

Carbonic anhydrase II deficiency is incorrect. This is a very rare condition and is associated with a mixed type 3 RTA picture. It would not explain nephrolithiasis and is not the most likely answer.

Topiramate use is incorrect. This is associated with type 2 RTA. You would not expect to see acidification of the urine as it is associated with type 2 RTA.

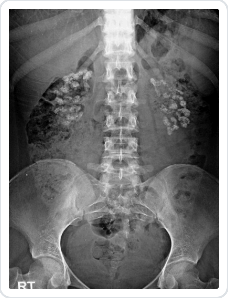
👍 👎 Discuss (5) Improve

Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

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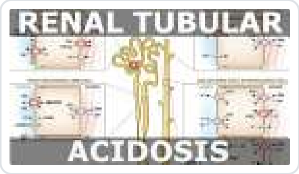
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A 62-year-old male, recently emigrated from India, presents with 5 day history of feeling generally unwell. His niece, who has accompanied him to hospital, denies a history of recent productive cough, diarrhoea or vomiting or dysuria. Her uncle had been gradually increasingly malaised over the past 5 days and not eating and drinking well. He has no known past medical history. On examination, he has dry mucous membranes and cool peripheries, his JVP is +1cm above the angle of Louis. Heart sounds, chest and abdomen are unremarkable. Urine dip and chest radiograph are awaited. His blood tests are as follows:

WBC	16 * 10^9/l
Neutrophils	14.8 * 10^9/l

Na^+	152 mmol/l
K^+	3.7 mmol/l
Urea	22 mmol/l
Creatinine	208 µmol/l
CRP	38 mg/l
Glucose	38 mmol/l
Ketones	2.8 mmol/l

Arterial blood gases:

pH 7.31
PaO2 20.2 kPa
PaCO2 3.0 kPa
Bicarbonate 16 mmol/l
Lactate 4 mmol/l

What is the unifying diagnosis?

○ Diabetic ketoacidosis (DKA) ✕

○ Lactic acidosis ✕

○ Hyperosmolar hyperglycaemic state (HHS) ✕

○ Urinary tract sepsis ✕

○ Chest sepsis ✕

Submit answer

Reference ranges ∨

Score: **56.3%**

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Arterial blood gases:

pH 7.31
PaO2 20.2 kPa
PaCO2 3.0 kPa
Bicarbonate 16 mmol/l
Lactate 4 mmol/l

What is the unifying diagnosis?

🟥	Diabetic ketoacidosis (DKA)	22%
	Lactic acidosis	6%
🟩	Hyperosmolar hyperglycaemic state (HHS)	70%
	Urinary tract sepsis	2%
	Chest sepsis	1%

This patient has presented with dehydration and non-specific symptoms and a diagnosis difficult to diagnose clinically. However, his biochemistry is diagnostic: calculation of his osmolality, (2[Na + K] + urea+ glucose) reveals an osmolality greater 371.4mosmol/kg. He is likely to present acutely with undiagnosed type 2 diabetes mellitus and a diagnosis of HHS, previously known as HONK.

There is no evidence to suggest uro or chest sepsis but an infectious underlying decompensating trigger should be considered with a prescription of broad spectrum antibiotics. Although lactate is mildly raised, this is likely secondary to intravascular dehydration and hypoperfusion of internal organs. Lactic acidosis alone does not account for the full biochemical picture. Ketones are present and the patient is mild acidotic. However, be aware that neither is sufficiently significant for a diagnosis of DKA.

👍 👎 Discuss (6) Improve

Next question

Hyperosmolar hyperglycaemic state ★

Hyperosmolar hyperglycaemic state (HHS) is a medical emergency that can be difficult to manage and has a significant associated mortality (up to 20%). Hyperglycaemia results in osmotic diuresis, severe dehydration, and electrolyte deficiencies. HHS typically presents in the elderly with type 2 diabetes mellitus (T2DM).

Pathophysiology

- hyperglycaemia → ↑ serum osmolality → osmotic diuresis → severe volume depletion

Precipitating factors

- intercurrent illness
- dementia
- sedative drugs

Clinical features

- whilst DKA presents within hours of onset, HHS comes on over many days, and consequently, the dehydration and metabolic disturbances may be more extreme
- consequences of volume loss
  - clinical signs of dehydration
  - polyuria
  - polydipsia
- systemic
  - lethargy
  - nausea and vomiting
- neurological
  - altered level of consciousness
  - focal neurological deficits
- haematological
  - hyperviscosity (may result in myocardial infarctions, stroke and peripheral arterial thrombosis)

There are no precise **diagnostic criteria** but the following are typically seen

- hypovolaemia
- marked hyperglycaemia (>30 mmol/L)
- significantly raised serum osmolarity (> 320 mosmol/kg)
  - can be calculated by: 2 * Na^+ + glucose + urea
- no significant hyperketonaemia (<3 mmol/L)
- no significant acidosis (bicarbonate > 15 mmol/l or pH > 7.3 - acidosis can occur due to lactic acidosis or renal impairment)

Management

- fluid replacement
    - fluid losses in HHS are estimated to be between 100 - 220 ml/kg
    - IV 0.9% sodium chloride solution
    - typically given at 0.5 - 1 L/hour depending on clinical assessment
    - potassium levels should be monitored and added to fluids depending on the level
- insulin
    - should not be given unless blood glucose stops falling while giving IV fluids
- venous thromboembolism prophylaxis
    - patients are at risk of thrombosis due to hyperviscosity

Complications

- vascular complications may occur due to hyperviscosity:
    - such as myocardial infarction
    - stroke

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Diabetes Societies 👍 20 👎 13

The management of the hyperosmolar hyperglycaemic state (HHS)

Suggest link

Report broken link

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A 70-year-old woman is brought to the emergency department due to confusion, dizziness and weakness. She has had several similar episodes in the past 3 months. Her past medical history includes type 2 diabetes mellitus of 20 years duration, hypertension, and hyperlipidaemia. Her diabetes is managed with metformin and glimepiride.

Her observations are taken as follows: temperature 37°C, blood pressure 110/70 mmHg, heart rate 90/min, and respiratory rate 16/min. Physical examination shows no abnormalities.

Blood tests are taken, and her finger-stick glucose is 2.7 mmol/L. Glucagon IM and 50 ml of 50% dextrose IV are given. She remains confused, and a repeat finger-stick glucose is 2.9 mmol/L. Serum insulin and C-peptide levels, drawn before dextrose administration, are elevated.

Which of the following is the next best step in the management of this patient?

- ○ Recheck insulin and C-peptide levels
- ○ Spiral computed tomography (CT) of the abdomen
- ○ Repeat glucagon IM
- ○ Observe for 30 minutes and recheck finger-stick glucose
- ○ Administer octreotide IM

Submit answer

Reference ranges

Score: **55.7%**

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Which of the following is the next best step in the management of this patient?

	Recheck insulin and C-peptide levels	4%
	Spiral computed tomography (CT) of the abdomen	15%
🟥	Repeat glucagon IM	13%
	Observe for 30 minutes and recheck finger-stick glucose	5%
🟩	Administer octreotide IM	63%

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Important for me Less important

This patient has symptomatic hypoglycaemia and elevated insulin and C-peptide levels, suggesting that the cause of her hypoglycaemia is excessive insulin secretion by her pancreas. Injectable insulins do not have C-peptide. There are two possible causes of excessive endogenous insulin secretion: insulinoma and sulphonylurea overdose. In this patient taking glimepiride, the latter is the more likely cause. In sulphonylurea overdoses, if the patient remains hypoglycaemic despite the infusion of sufficient glucose, consider administration of octreotide.

Sulphonylurea overdose is treated with bolus dextrose solution. Most patients recover after this intervention. However, since sulphonylureas cause the pancreas to continue releasing insulin, patients are at an increased risk of persistent or recurrent hypoglycaemia. In such patients, 50 to 100 mcg octreotide is administered intramuscularly or subcutaneously. Octreotide is a somatostatin analogue that inhibits insulin release from pancreatic beta-islet cells.

Rechecking insulin and C-peptide levels is inappropriate in this case and would lead to unnecessary delays in management. This patient with symptomatic hypoglycaemia needs urgent treatment.

While insulinoma is a possible cause of raised insulin and C-peptide levels, it is rare and quite unlikely in this patient. Moreover, getting a CT scan would delay the management of her hypoglycaemia.

Repeating glucagon administration may control the hypoglycaemia, but it will not address the underlying cause of persistent hypoglycaemia. The best next step is to administer octreotide.

This patient has symptomatic hypoglycaemia in the setting of sulphonylurea overdose. Observing for 30 minutes would worsen her hypoglycaemia and may lead to coma or death.

👍 👎 💬 Discuss (5) Improve

Next question

Hypoglycaemia ★

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
  - causes exaggerated insulin secretion
  - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia

- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later
- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic) neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features

- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
  - Sweating
  - Shaking
  - Hunger
  - Anxiety
  - Nausea
- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
  - Weakness
  - Vision changes

  - Confusion
  - Dizziness
- Severe and uncommon features of hypoglycaemia include:
  - Convulsion
  - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
  - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form

    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
    - If the patient is alert, a quick-acting carbohydrate may be given (as above)
    - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
    - Alternatively, intravenous 20% glucose solution may be given through a large vein

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.1%**

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A 54-year-old man presented to endocrine clinic with gradually worsening low mood, malaise, and reduced exercise capacity. . He was operated on previously for a non-functioning pituitary adenoma (NFPA) resulting in partial anterior hypopituitarism, and had been on thyroxine and hydrocortisone replacement therapy. On examination, he had evidence of central adiposity. He was known to have dyslipidaemia, epilepsy and ischaemic heart disease (IHD) and so an insulin tolerance test was contraindicated.

Investigations

9a.m cortisol	415nmol/l
IGF-1	8nmol/l (16 - 118)
FT4	15.4nmol/l (11.5 - 22.7)
TSH	0.03mU/l (0.35 - 5.5)

Which one of the following tests is most appropriate in his case to confirm the diagnosis of adult GH deficiency?

- ○ Domperidone test ×
- ○ Growth hormone levels ×
- ○ Growth hormone releasing hormone (GHRH)-arginine stimulation test ×
- ○ Insulin-like growth factor- binding protein measurement ×
- ○ Glucose tolerance test ×

Submit answer

Reference ranges

Score: **55.1%**

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Question 90 of 342 ✓

A 54-year-old man presented to endocrine clinic with gradually worsening low mood, malaise, and reduced exercise capacity. . He was operated on previously for a non-functioning pituitary adenoma (NFPA) resulting in partial anterior hypopituitarism, and had been on thyroxine and hydrocortisone replacement therapy. On examination, he had evidence of central adiposity. He was known to have dyslipidaemia, epilepsy and ischaemic heart disease (IHD) and so an insulin tolerance test was contraindicated.

Investigations

9a.m cortisol	415nmol/l
IGF-1	8nmol/l (16 - 118)
FT4	15.4nmol/l (11.5 - 22.7)
TSH	0.03mU/l (0.35 - 5.5)

Which one of the following tests is most appropriate in his case to confirm the diagnosis of adult GH deficiency?

Domperidone test	1%
Growth hormone levels	3%
[✓ Growth hormone releasing hormone (GHRH)-arginine stimulation test]	40%
Insulin-like growth factor- binding protein measurement	28%
Glucose tolerance test	27%

Arginine-GHRH stimulation test is employed to assess GH secretion in patients with ischemic heart disease or seizures where insulin tolerance test will be inappropriate

Important for me Less important

The clinical features of growth hormone deficiency (GHD) are non-specific and include lethargy, low mood, poor quality of life, loss of muscle mass, and central adiposity. A low IGF-1 in such a clinical context may point towards GHD, which needs to be confirmed with dynamic tests for GH secretion. Insulin tolerance test (ITT) is considered the gold standard for assessing GH secretion, although it needs to be conducted carefully in a closely-monitored space due to the risks associated with hypoglycaemia.

ITT is contra-indicated in patients with seizures or ischaemic heart disease. As a result, an alternative test such as arginine-GHRH stimulation test is employed (as in this clinical scenario where the patient has IHD). About 30-40% of patients with GHD may have a normal IGF-1 level. IGF-1 levels are influenced by age, time of onset of GHD, and degree of hypopituitarism. Domperidone test is used in the evaluation of hyperprolactinemia and macroprolactinaemia associated disorders while glucose tolerance test with measurement of IGF-1 levels is used in the evaluation of suspected acromegaly.

👍 👎 Discuss (3) Improve

Next question

Hypopituitarism ★

Hypopituitarism describes the inadequate production of one or more of the hormones secreted by the pituitary gland.

Causes

- compression of the pituitary gland by non-secretory pituitary macroadenoma (most common)
- pituitary apoplexy
- Sheehan's syndrome: postpartum pituitary necrosis secondary to a postpartum haemorrhage
- hypothalamic tumours e.g. craniopharyngioma
- trauma

- iatrogenic irradiation
- infiltrative e.g. hemochromatosis, sarcoidosis

The clinical features depend on which hormones are deficient:
- low ACTH
  - tiredness
  - postural hypotension
- low FSH/LH
  - amenorrhoea
  - infertility
  - loss of libido
- low TSH
  - feeling cold
  - constipation
- low GH
  - if occurs during childhood then short stature
- low prolactin
  - problems with lactation
- there may also be features suggestive of the underlying causes
  - pituriary macroadenoma → bitemporal hemianopia
  - pituitary apoplexy → sudden, severe headache

Investigations
- hormone profile testing
- imaging

Management
- treatment of any underlying cause (e.g. surgical removal of pituitary macroadenoma)
- replacement of deficient hormones

+ Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Media

HYPOPITUITARISM
Osmosis.org

Hypopituitarism

Osmosis - YouTube 👍 5 👎 0

Report broken media

Score: **55.6%**

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A frail 82-year-old gentleman was brought in by his daughter, who found him on the floor in his flat. He had tripped in a mechanical and had been unable to get back up, lying on the floor for the past 3 days. On examination, he appears extremely dehydrated but has no specific focal weakness, systemic examination is unremarkable. He has sustained no musculoskeletal injuries. His blood tests are as follows:

Na^+	168 mmol/l
K^+	6.0 mmol/l
Urea	24 mmol/l
Creatinine	260 µmol/l (baseline 107 three months ago)
Creatinine kinase	11,000 mmol/l

ECG shows normal sinus rhythm at 99/ minute.

You diagnose him with rhabdomyolysis and an acute kidney injury, likely of a pre-renal cause. Intravenous fluid rehydration is initiated with intravenous 5% dextrose. You ask your colleague to check the patient's blood tests in 12 hours.

What is the aim of correcting the patient's hypernatraemia?

○ Reduce blood sodium to under 145 mmol/l as quickly as possible ×

○ Reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l ×

○ Reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l ×

○ Aim for blood sodium above 145 mmol/l ×

○ Blood sodium does not require monitoring if intravenous fluids is running, CK falling and renal function improving ✗

Submit answer

Reference ranges

Score: **55.6%**

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Question 91 of 342 ✓

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What is the aim of correcting the patient's hypernatraemia?

Reduce blood sodium to under 145 mmol/l as quickly as possible	1%
[✓ Reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l]	75%
Reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l	16%
Aim for blood sodium above 145 mmol/l	0%

Blood sodium does not require monitoring if intravenous fluids is running, CK falling and renal function improving 8%

The correct answer is to **reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l**. In cases of hypernatraemia, it is important to correct the patient's sodium levels gradually to avoid complications such as cerebral oedema and central pontine myelinolysis. Aiming for a reduction rate of 0.5mmol/hr ensures that the correction is slow and controlled, allowing the body to adapt safely.

The first option, **reduce blood sodium to under 145 mmol/l as quickly as possible**, is incorrect because rapidly correcting hypernatraemia can lead to severe neurological complications such as osmotic demyelination syndrome (central pontine myelinolysis). It is crucial to correct sodium levels slowly and cautiously.

The second incorrect option, **reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l**, suggests a faster rate of correction than recommended. This approach also increases the risk of complications, particularly in elderly patients who may have reduced physiological reserve and increased vulnerability to rapid changes in serum electrolyte concentrations.

The fourth option, **aim for blood sodium above 145 mmol/l**, contradicts the goal of treating hypernatraemia. A normal serum sodium level ranges from approximately 135-145 mmol/l; thus, maintaining a level above this range would not address the patient's underlying issue.

Finally, the last option, **blood sodium does not require monitoring if intravenous fluids are running, CK falling and renal function improving**, is incorrect because regular monitoring of blood sodium levels during treatment for rhabdomyolysis and acute kidney injury is essential. Monitoring allows clinicians to assess response to therapy, adjust fluid management as needed, and detect potential complications early.

👍 👎 💬 Discuss (7) Improve

Next question

Hypernatraemia

Causes of hypernatraemia

- dehydration
- osmotic diuresis e.g. hyperosmolar non-ketotic diabetic coma
- diabetes insipidus
- excess IV saline

Hypernatraemia should be corrected with great caution. Although brain tissue can lose sodium and potassium rapidly, lowering of other osmolytes (and importantly water) occurs at a slower rate, predisposing to cerebral oedema, resulting in seizures, coma and death1. Although there are no clinical guidelines by NICE or Royal College of Physicians at present, it is generally accepted that a rate of no greater than 0.5 mmol/hour correction is appropriate[1].

1. Reynolds RM, Padfield PL, Seckl JR; Disorders of sodium balance. BMJ. 2006 Mar 25;332(7543):702-5.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **56%**

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K^+	5.1 mmol/l
Urea	4.7 mmol/l
Creatinine	81 µmol/l
Calcium	1.9 mmol/l
Random glucose	3.9 mmol/l

What is the most likely diagnosis?

○ Type II polyglandular autoimmune syndrome ×

○ Thymoma ×

- ○ Type 1 polyglandular autoimmune syndrome ×
- ○ Type III polyglandular autoimmune syndrome ×
- ○ HIV ×

Submit answer

Reference ranges

Score: **56%**

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What is the most likely diagnosis?

Type II polyglandular autoimmune syndrome	32%
Thymoma	1%

Type 1 polyglandular autoimmune syndrome	58%
Type III polyglandular autoimmune syndrome	3%
HIV	6%

The most likely diagnosis is type 1 polyglandular autoimmune syndrome. This autosomal recessive syndrome is a subtype of autoimmune polyendocrine syndrome, whereby a number of endocrine glands dysfunction. The patient's oral candidiasis is caused by a mild immune deficiency and hyposplenism. Furthermore, the patient has hypocalcaemia, caused by autoimmune dysfunction of the parathyroid gland and hypoglycaemia with hypotension, caused by autoimmune dysfunction of the adrenal gland.

👍 👎 Discuss (7) Improve

Next question

Autoimmune polyendocrinopathy syndrome ★

Addison's disease (autoimmune hypoadrenalism) is associated with other endocrine deficiencies in approximately 10% of patients. There are two distinct types of autoimmune polyendocrinopathy syndrome (APS), with type 2 (sometimes referred to as Schmidt's syndrome) being much more common.

APS type 2 has a polygenic inheritance and is linked to HLA DR3/DR4. Patients have Addison's disease plus either:
- type 1 diabetes mellitus
- autoimmune thyroid disease

APS type 1 is occasionally referred to as Multiple Endocrine Deficiency Autoimmune Candidiasis (MEDAC). It is a very rare autosomal recessive disorder caused by mutation of AIRE1 gene on chromosome 21

Features of APS type 1 (2 out of 3 needed)

- chronic mucocutaneous candidiasis (typically first feature as young child)
- Addison's disease
- primary hypoparathyroidism

Vitiligo can occur in both types

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

AUTOIMMUNE
POLYGLANDULAR
SYNDROME TYPE 1

Autoimmune polyglandular syndrome type 1

Osmosis - YouTube 👍 5 👎 0

Report broken media

Score: **55.4%**

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Osmosis.org

Autoimmune polyglandular syndrome type 1

Osmosis - YouTube 👍 5 👎 0

Report broken media

Score: **55.4%**

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A 27-year-old female was seen in the general medicine outpatient clinic following a referral by her GP. She had presented to her GP feeling generally tired for the last few months. Her GP organised a blood screen revealing no abnormalities except the presence of a potassium level of 2.8 mmol/l. Her GP subsequently repeated the test four weeks later revealing a level of 2.6 mmol/l, leading to the referral to the clinic. Other than the tiredness she was well in herself, denying all other symptoms. She denied specifically the presence of any cardiovascular and muscular symptoms and had never collapsed. Her past medical history was unremarkable and she was taking no prescribed medication. There was no family history of note.

Examination revealed a well 27-year-old lady. Her blood pressure was 108/78 mmHg, heart rate 82 bpm and BMI 23kg/m². Examination of her cardiovascular and respiratory systems revealed the presence of normal heart sounds, a JVP of 3cm and warm well perfused peripheries. Examination of her gastrointestinal and neurological systems was unremarkable.

Investigations conducted at the clinic revealed the following results:

Hb	122 g/l
Platelets	242 * 10^9/l
WBC	7.8 * 10^9/l

Na^+	132 mmol/l
K^+	2.5 mmol/l
Urea	7.2 mmol/l
Creatinine	68 µmol/l
Bicarbonate	37 mmol/l

Serum renin	824 (NR 100-500 pmol/l)

Serum aldosterone	82 (NR 55-250 pmol/l)
TSH	1.2 mu/l

24 hour urine result:

Na+	28 (NR <20mmol/L if hyponatraemia)
K+	45 (NR <10mmol/l if hypokalaemic)
Calcium	0.8 (NR <7.5 mmol/24hrs)

What is the most likely diagnosis?

○ Addison's disease ×

○ Gitelman's syndrome ×

○ Conn's disease ×

○ Bartter's syndrome ×

○ Laxative abuse ×

Submit answer

Reference ranges ⌄

Score: **55.4%**

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What is the most likely diagnosis?

Addison's disease	5%
[✓ Gitelman's syndrome]	57%
Conn's disease	7%
Bartter's syndrome	23%
Laxative abuse	8%

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

Important for me Less important

Gitelman's syndrome is an autosomal recessive disorder resulting in a normotensive hypokalaemic metabolic alkalosis with hypocalciuria and is often accompanied with hypomagnesaemia. The defect is in the thiazide-sensitive sodium chloride symporter within the distal convoluted tubules, in contrast to Bartter's syndrome which presents in the same way but with hypercalciuria owing to a defect within the ascending loop of Henle. Patients with both conditions are often asymptomatic or may complain of fatigue, cramps and weakness. Conn's disease is associated with hypertension and in this instance the aldosterone level is normal with an elevated renin, making this diagnosis unlikely. Both Addison's disease and laxative abuse are associated with a metabolic acidosis; in Addison's serum potassium also tends to be elevated. The best answer is, therefore, Gitelman's syndrome.

Discuss (9) Improve

Next question

Gitelman's syndrome

Gitelman's syndrome is due to a defect in the thiazide-sensitive Na^+ Cl- transporter in the distal convoluted tubule.

Features
- normotension
- hypokalaemia
- hypocalciuria
- hypomagnesaemia
- metabolic alkalosis

Diagnosis
- genetic testing for mutations in the SLC12A3 gene

Management
- oral potassium and magnesium supplementation
- potassium-sparing diuretics may be used if still hypokalaemic/symptomatic

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.9%**

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A 32-year-old patient is due to be discharged from hospital. He has had his third admission for DKA in the last two years since being diagnosed with type 1 diabetes mellitus. He has come off his fixed-rate IV insulin and started to eat and drink again, and has restarted his long-acting insulin detemir (Levemir) and short-acting insulin aspart (Novorapid) as his pre-admission plan. His DKA was most likely provoked by the patient drinking alcohol and not taking his insulin. This is similar to previous admissions, but he only drinks two bottles of beer a week, but occasionally binge drinks. He has no other co-morbidities and takes no other medications. What is the most appropriate suggestion to avoid further admissions with DKA?

- ○ Start metformin ×
- ○ Change insulin detemir (Levemir) to insulin degludec (Tresiba) ×
- ○ Change insulin detemir (Levemir) to insulin aspart biphasic (Novomix 30) ×
- ○ Increase dose in insulin aspart (Novorapid) by 20% ×
- ○ Advise reduction in carbohydrates in diet ×

Submit answer

Reference ranges ⌄

Score: **55.9%**

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Start metformin	8%
Change insulin detemir (Levemir) to insulin degludec (Tresiba)	40%
Change insulin detemir (Levemir) to insulin aspart biphasic (Novomix 30)	29%
Increase dose in insulin aspart (Novorapid) by 20%	10%
Advise reduction in carbohydrates in diet	12%

A patient with recurrent admissions for DKA can be started on degludec to reduce readmission rate

Important for me Less important

This is a patient with type 1 diabetes with recurrent DKA secondary to a combination of missing doses of insulin and binge drinking alcohol. The most appropriate and likely to be successful strategy is to change Levemir to Degludec. Degludec has a much higher half-life than Levemir and therefore maintains a basal insulin level when the patient omits or forgets doses. This can prevent DKA. Metformin is not indicated for this patient. Insulin aspart biphasic (Novomix 30) would allow reduction in the number of insulin injections. Increasing the dose of insulin aspart (Novorapid) is unlikely to prvent DKA for this patient as the fundamental issue is missing doses, and the short-term nature of the insulin aspart (Novorapid) is unlikely to provide any insulin cover for further omitted doses. Patients with type 1 diabetes can also benefit from DAFNE, a course which helps patients adjust their insulin doses to what they are eating and the patient should be offered this as an educational tool as well to improve control.

👍 👎 💬 Discuss (6) Improve

Next question

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	Joint British Diabetes Societies (2013)
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids
  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

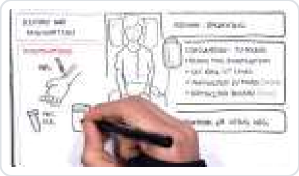
Links

Joint British Inpatient Diabetes Societies Inpatient Care Group 👍 33 👎 23

The Management of Diabetic ketoacidosis in adults

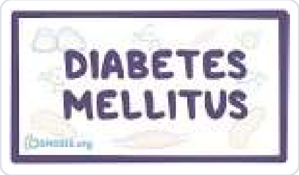
Suggest link Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **56.4%**

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Question 95 of 342

A 65-year-old man who is known to have type 2 diabetes mellitus presents for advice. He is a Muslim and is considering fasting for Ramadan. His diabetes is currently controlled with a combination of diet and metformin 500mg tds. Looking at his records the last HbA1c was 6.4% (46 mmol/mol). If he decides to fast during Ramadan, what is the most appropriate advice to give regarding his metformin?

- ○ Metformin should be stopped ✕
- ○ Metformin 1.5g after sunset ✕
- ○ Metformin 500mg before sunrise, 1g after sunset ✕
- ○ Metformin 500mg after sunset ✕
- ○ Metformin 1g before sunrise, 500mg after sunset ✕

Submit answer

Reference ranges

Score: **56.4%**

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A 65-year-old man who is known to have type 2 diabetes mellitus presents for advice. He is a Muslim and is considering fasting for Ramadan. His diabetes is currently controlled with a combination of diet and metformin 500mg tds. Looking at his records the last HbA1c was 6.4% (46 mmol/mol). If he decides to fast during Ramadan, what is the most appropriate advice to give regarding his metformin?

Metformin should be stopped	2%
Metformin 1.5g after sunset	2%
Metformin 500mg before sunrise, 1g after sunset	83%
Metformin 500mg after sunset	3%
Metformin 1g before sunrise, 500mg after sunset	10%

During Ramadan, one-third of the normal metformin dose should be taken before sunrise and two-thirds should be taken after sunset

Important for me Less important

The correct answer is **Metformin 500mg before sunrise, 1g after sunset**. This is because during Ramadan, individuals who fast are required to refrain from consuming food and drink from sunrise to sunset. In order to maintain adequate glycaemic control while minimizing the risk of hypoglycaemia, it is recommended that metformin be taken in a divided dose - a smaller dose before sunrise (pre-dawn meal or Suhoor) and a larger dose after sunset (evening meal or Iftar). This allows for better distribution of the medication throughout the day while still adhering to the fasting requirements.

Metformin should be stopped is incorrect because stopping metformin abruptly may result in poor glycaemic control and increased risk of hyperglycaemia. It is important for patients with diabetes to continue their medications during Ramadan, with appropriate adjustments made under medical supervision.

Metformin 1.5g after sunset is not the best option because taking the entire daily dose at once can increase the risk of gastrointestinal side effects and may not provide optimal glycaemic control throughout the day. Dividing the dose as mentioned earlier helps maintain better blood glucose levels during fasting hours.

Metformin 500mg after sunset is also incorrect because this dosing regimen significantly reduces the total daily dose of metformin compared to his current regimen, which may lead to inadequate glycaemic control. Additionally, it does not account for any pre-dawn medication intake.

Finally, **Metformin 1g before sunrise, 500mg after sunset** is not ideal as it provides a higher dose before sunrise than necessary. The recommended approach is to take a smaller dose before sunrise and a larger one after sunset in order to minimize potential hypoglycaemia during fasting hours while maintaining adequate glycaemic control.

👍 👎 Discuss (9) Improve

Next question

Diabetes mellitus: Ramadan ★

We know that type 2 diabetes mellitus is more common in people of Asian ethnicity and a significant proportion of those patients in the UK will be Muslim. The BMJ published an excellent and comprehensive review of this issue in 2010[1].

It is important that we can give appropriate advice to Muslim patients to allow them safely observe their fast. This is particularly important from 2014 as Ramadan is due to fall in the long days of the summer months for several years henceforth.

Clearly it is a personal decision whether a patient decides to fast. It may however be worthwhile exploring the fact that people with chronic conditions are exempt from fasting or may be able to delay fasting to the shorter days of the winter months. It is however known that many Muslim patients with diabetes do not class themselves as having a chronic/serious condition which should exempt them from fasting. Around 79% of Muslim patients with type 2 diabetes mellitus fast Ramadan[2].There is an excellent patient information leaflet from Diabetes UK and the Muslim Council of Britain which explores these options in more detail.

If a patient with type 2 diabetes mellitus does decide to fast:

- they should try and and eat a meal containing long-acting carbohydrates prior to sunrise (Suhoor)
- patients should be given a blood glucose monitor to allow them to check their glucose levels, particularly if they feel unwell
- for patients taking metformin the expert consensus is that the dose should be split one-third before sunrise (Suhoor) and two-thirds after sunset (Iftar)
- expert consensus also recommends switching once-daily sulfonylureas to after sunset. For patients taking twice-daily preparations such as gliclazide it is recommended that a larger proportion of the dose is taken after after sunset
- no adjustment is needed for patients taking pioglitazone

1. Management of people with diabetes wanting to fast during Ramadan BMJ 2010;340:c3053
2. Salti I et al. Results of the Epidemiology of Diabetes and Ramadan (EPIDIAR) study. Diabetes Care 2004;27:2306-11.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Diabetes Care 👍 22 👎 6

Recommendations for Management of Diabetes During Ramadan

Diabetes UK and Muslim Council 👍 25 👎 4

Patient information leaflet on the management of diabetes during Ramadan

Suggest link

Report broken link

Score: **56.8%**

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Back to top

A 23-year-old female presents with worsening acne and a marked increase in the development of body and facial hair which she finds very distressing. She is also overweight and is markedly stressed by her physical appearance and the development of stretch marks over her abdomen. She has tried multiple hair removal techniques with only mild success.

On examination, she has a body mass index of 28 kg/m², coarse hair over the anterior and posterior part of her chest and under her chin. Her Blood Pressure is 135/90mmHg.

Her lab results are as follows:

9:00 am Cortisol	345 nmol/l (170 700 nmol/l)
LH	17 iU/l (1 20 iU/l)
Basal FSH	7.1 iU/l (1.0 8.8 iU/l)
DHEAS	545 µg/dl (31 228 µg/dl)
Prolactin	160 mU/l (<360 mU/l)
17 OH Progesterone	1025 ng/dl (<80 ng/dl)
Testosterone	3.9 nmol/l (0.9 3.1 nmol/l)

Ultrasound abdomen and pelvis reveals two cysts in the right ovary.

Which of the following is the most appropriate treatment option for her condition?

○ Combined oral contraceptive pill ×

○ Finasteride ×

○ Surgical resection of the ovarian cysts ×

○ Reverse circadian rhythm steroids ✗

○ Metformin in combination with spironolactone ✗

Submit answer

Reference ranges

Score: **56.8%**

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Prolactin	160 mU/l (<360 mU/l)
17 OH Progesterone	1025 ng/dl (<80 ng/dl)
Testosterone	3.9 nmol/l (0.9 3.1 nmol/l)

Ultrasound abdomen and pelvis reveals two cysts in the right ovary.

Which of the following is the most appropriate treatment option for her condition?

Combined oral contraceptive pill	44%
Finasteride	12%
Surgical resection of the ovarian cysts	3%

🟩	Reverse circadian rhythm steroids	13%
🟥	Metformin in combination with spironolactone	28%

The diagnosis in this scenario is non-classical congenital adrenal hyperplasia which manifests in adolescence/adulthood. It is caused by a deficiency of the enzyme 21 hydroxylase in the steroid biosynthetic pathway. The result is a shift in the production of steroid hormones towards the androgenic pathway. Since cortisol secretion is reduced, feedback leads to increased ACTH production and resultant hyperplasia of the adrenals. The level of the compounds that are formed prior to the action of 21 hydroxylase is increased, therefore levels of 17 hydroxyprogesterone are elevated. Due to excessive androgen production, there is virilization and hirsutism.

Treatment involves steroids given in reverse circadian rhythm, i.e. a higher dosage at night and a lower dose in the morning.

The rationale behind this approach is due to the pathophysiology of CAH. The adrenal hyperplasia and the over-secretion of adrenal androgens are due to excessive ACTH production. When steroids are given in higher doses at night, ACTH is suppressed and the normal physiological steroid peak in the morning is also reduced.

Cysts in the ovaries are a common finding on routine ultrasound and do not necessarily represent polycystic ovarian syndrome.

For further review please see http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2266964/ entitled Approach to the Adult with Congenital Adrenal Hyperplasia due to 21-Hydroxylase Deficiency.

Discuss (19) | Improve

Next question

Congenital adrenal hyperplasia: features

21-hydroxylase deficiency features

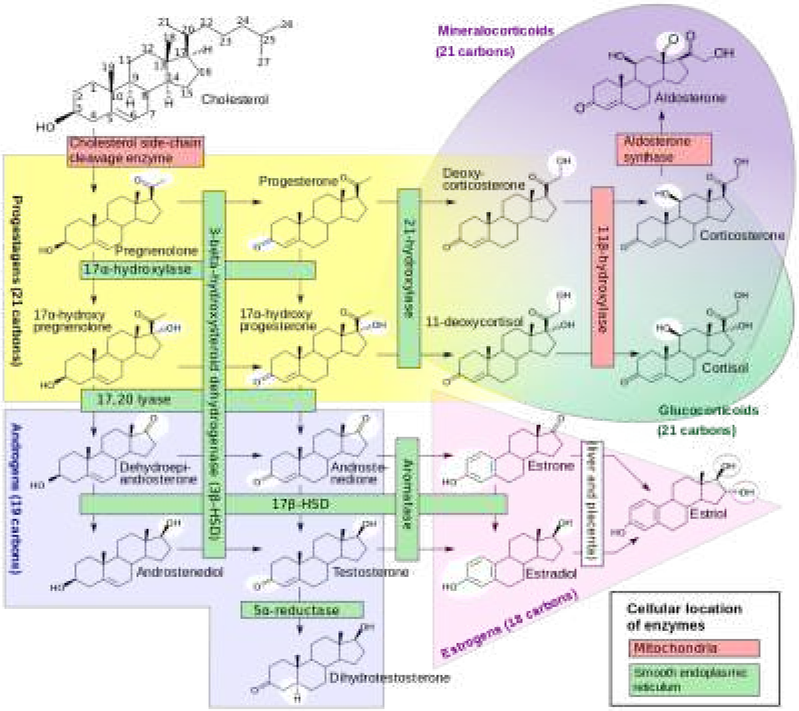
- virilisation of female genitalia
- precocious puberty in males
- 60-70% of patients have a salt-losing crisis at 1-3 wks of age

11-beta hydroxylase deficiency features

- virilisation of female genitalia
- precocious puberty in males
- hypertension
- hypokalaemia

17-hydroxylase deficiency features

- non-virilising in females
- inter-sex in boys
- hypertension


Cholesterol
Cholesterol side-chain cleavage enzyme
Progestagens (21 carbons)
Pregnenolone
Progesterone
17α-hydroxylase
3-beta-hydroxysteroid dehydrogenase (3β-HSD)
21-hydroxylase
17α-hydroxy pregnenolone
17α-hydroxy progesterone
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Androgens (19 carbons)
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liver and placenta
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Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

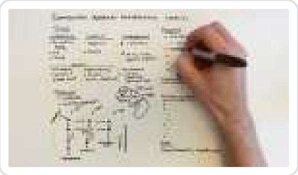
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 👍 13 👎 0

Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 👍 10 👎 0

Report broken media

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Question 97 of 342

A 52-year-old man presents to the endocrine clinic with a 1-month history of polyuria and polydipsia. He has no significant past medical history and takes no regular medications.

Blood results:

Na^+	156 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	162 µmol/L	(55 - 120)

Osmolarity studies after water deprivation are as follows:

Urine osmolarity	105 mmol/L	(300-900)
Plasma osmolarity	332.5 mOsm/kg	(275 - 295)

Results following administration of desmopressin:

Urine osmolarity	322 mmol/L	(300-900)
Plasma osmolarity	324.5 mOsm/kg	(275 - 295)

What is the most likely diagnosis?

- ○ Complete cranial diabetes insipidus ×
- ○ Nephrogenic diabetes insipidus ×
- ○ Partial cranial diabetes insipidus ×
- ○ Primary polydipsia ×

○ Syndrome of inappropriate antidiuretic hormone release ×

Submit answer

Reference ranges ⌄

Score: **56.3%**

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A 52-year-old man presents to the endocrine clinic with a 1-month history of polyuria and polydipsia. He has no significant past medical history and takes no regular medications.

Blood results:

Na^+	156 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	162 µmol/L	(55 - 120)

Osmolarity studies after water deprivation are as follows:

Urine osmolarity	105 mmol/L	(300-900)
Plasma osmolarity	332.5 mOsm/kg	(275 - 295)

Results following administration of desmopressin:

Urine osmolarity	322 mmol/L	(300-900)
Plasma osmolarity	324.5 mOsm/kg	(275 - 295)

What is the most likely diagnosis?

🟩	Complete cranial diabetes insipidus	37%
🟥	Nephrogenic diabetes insipidus	21%
	Partial cranial diabetes insipidus	37%
	Primary polydipsia	3%

Syndrome of inappropriate antidiuretic hormone release 1%

Water deprivation test: cranial DI
- urine osmolality after fluid deprivation: low
- urine osmolality after desmopressin: high

Important for me Less important

Complete cranial diabetes insipidus is correct. The water deprivation test confirms a diagnosis of diabetes insipidus (DI) due to a failure of the urine to concentrate with dehydration (urine osmolarity <300 mmol/L). Following administration of desmopressin there is a >50% rise in urine osmolarity confirming the diagnosis of cranial DI.

Partial cranial diabetes insipidus is incorrect. This subtype of cranial DI is confirmed when the patient's symptoms resolve with desmopressin however the urine osmolarity fails to meet the diagnostic criteria (e.g. >50% increase).

Nephrogenic diabetes insipidus is incorrect. This condition is characterised by the failure of the urine to concentrate with desmopressin.

Primary polydipsia is incorrect. This condition is characterised by a normal physiological response to water deprivation e.g. the urine will concentrate (>600 mmol/L).

Syndrome of inappropriate antidiuretic hormone release is incorrect. Syndrome of inappropriate antidiuretic hormone release (SIADH) is associated with hyponatraemia, a decreased plasma osmolarity (<275 mOsm/kg), a relatively low urine osmolarity (< 100 mOsm/kg), and a high urinary sodium (> 40 mmol/L).

👍 👎 Discuss (6) Improve

Next question

Water deprivation test

The water deprivation test is designed to help evaluate patients who have polydipsia.

Method

- prevent patient drinking water
- ask the patient to empty their bladder
- hourly urine and plasma osmolalities

	Starting plasma osm.	Final urine osm.	Urine osm. post-DDAVP
Normal	Normal	> 600	> 600
Psychogenic polydipsia	Low	> 400	> 400
Cranial DI	High	< 300	> 600
Nephrogenic DI	High	< 300	< 300

+ Q 123

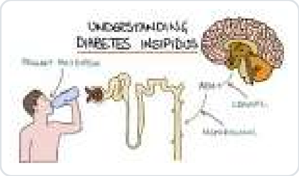
Next question

Textbooks

High-yield textbook

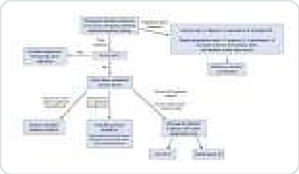
Extended textbook

Media


UNDERSTANDING DIABETES INSIPIDUS
ADH

Understanding Diabetes Insipidus

Zero to Finals - YouTube 👍 3 👎 0



An Approach to Polyuria

Strong Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **55.7%**

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A 47-year-old builder presented with paraesthesia in both hands which was worse at night. His hands felt swollen, although they were not painful, and he had needed to buy a larger pair of work gloves. When at work he found that his hands felt weak. Over the past six months, he had been experiencing urinary frequency, fatigue and increased thirst.

He had a past medical history of obesity and hypertension and his brother had type II diabetes mellitus. His only medication was ramipril. He was a heavy smoker with a 20 pack year history.

On examination of the arms, there was weakness of thumb abduction bilaterally and diminished sensation over the radial three and a half digits. Percussion over the palmar aspect of the wrist reproduced the paraesthesia he described on presentation. On examination of the chest and abdomen, there were areas of pigmentation in both axillae and striae over the abdomen. He had a protuberant abdomen and an elevated body mass index (BMI).

Which of the following investigations is most likely to be diagnostic?

- ○ Magnetic Resonance Imaging of the Pituitary and visual field testing ×
- ○ Fasting glucose on three occasions, glycosylated haemoglobin (HbA1c) and a 9am cortisol measurement ×
- ○ Growth hormone measurement and dexamethasone suppression test ×
- ○ Nerve conduction studies and electromyogram (EMG) ×
- ○ Oral glucose tolerance test with serum glucose, IGF-1 and growth hormone measurements ×

Submit answer

Reference ranges ⌄

Score: **55.7%**

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Which of the following investigations is most likely to be diagnostic?

Magnetic Resonance Imaging of the Pituitary and visual field testing	11%
Fasting glucose on three occasions, glycosylated haemoglobin (HbA1c) and a 9am cortisol measurement	8%
Growth hormone measurement and dexamethasone suppression test	9%
Nerve conduction studies and electromyogram (EMG)	2%
Oral glucose tolerance test with serum glucose, IGF-1 and growth hormone measurements	70%

This gentleman has bilateral carpal tunnel syndrome with an underlying diagnosis of acromegaly.

The features in the question which point to the diagnosis of acromegaly include the presence of a recent increase in hand size and known complications of acromegaly such as diabetes mellitus (polyuria and polydipsia), acanthosis nigricans (pigmentation in the axillae) and carpal tunnel syndrome.

The increased BMI and striae could be indicative of underlying Cushing's disease, but in this case, this were red herrings.

The diagnosis of acromegaly is made when there is a failure to suppress the release of growth hormone during an oral glucose tolerance test. This test also allows the diagnosis of diabetes mellitus to be made. Due to the diurnal variation in growth hormone levels, a random measurement is not helpful in making a diagnosis.

Although an MRI scan of the pituitary and visual field testing are very important investigations and can confirm the presence of a pituitary adenoma they will not confirm or exclude a diagnosis of acromegaly.

A fasting glucose and HbA1c measurement may aid the diagnosis of diabetes mellitus but not acromegaly.

A 9 am cortisol and dexamethasone suppression test could be used to investigate Cushing's syndrome and therefore are not relevant here.

Nerve conduction studies and electromyography would confirm the diagnosis of carpal tunnel syndrome but not the underlying cause, i.e. acromegaly.

👍 👎 Discuss (9) Improve

Next question

Acromegaly: investigations ★

Growth hormone (GH) levels vary during the day and are therefore not diagnostic.

Serum IGF-1 levels have now overtaken the oral glucose tolerance test (OGTT) with serial GH measurements as the first-line test. The OGTT test is recommended to confirm the diagnosis if IGF-1 levels are raised.

The Endocrine Society guidelines suggest the following:

> *1.1 We recommend measurement of IGF-1 levels in patients with typical clinical manifestations of acromegaly, especially those with acral and facial features.*
> ...
> *1.5 In patients with elevated or equivocal serum IGF-1 levels, we recommend confirmation of the diagnosis by finding lack of suppression of GH to < 1 μg/L following documented hyperglycemia during an oral glucose load.*

Serum IGF-1 may also be used to monitor disease

Oral glucose tolerance test
- in normal patients GH is suppressed to < 2 mu/L with hyperglycaemia
- in acromegaly there is no suppression of GH
- may also demonstrate impaired glucose tolerance which is associated with acromegaly

A pituitary MRI may demonstrate a pituitary tumour.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 14 👎 3

2014 Acromegaly guidelines

Suggest link

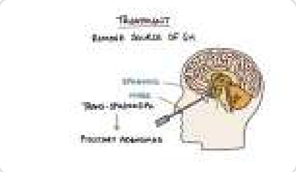
Report broken link

Media

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 1 👎 0


TREATMENT
REMOVE SOURCE OF GH

Understanding Acromegaly

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **56.1%**

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Question 99 of 342

A 59-year-old man presents to the medical clinic. He has a past history of type 2 diabetes mellitus, gout, obesity and cholecystectomy. He takes metformin and allopurinol. He has no allergies as far as he is aware. His total cholesterol is 4.8mmol/mol. He is concerned that his brother recently had a heart attack and was wondering if there is any medication that he should take to further reduce his risk of heart attacks. He smokes roughly five cigarettes per day, of which he has been trying to reduce. His blood pressure is 132/71mmHg. What is the most appropriate action?

- ○ Start atorvastatin 20mg ON ✕
- ○ Start atorvastatin 80mg ON ✕
- ○ Start aspirin 75mg OD ✕
- ○ Start clopidogrel 75mg OD ✕
- ○ Start ezetimibe 10mg OD ✕

Submit answer

Reference ranges

Score: **56.1%**

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Question 99 of 342 ✗

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🟩	Start atorvastatin 20mg ON	84%
🟥	Start atorvastatin 80mg ON	11%
	Start aspirin 75mg OD	4%
	Start clopidogrel 75mg OD	1%
	Start ezetimibe 10mg OD	1%

Patients with T2DM should not be routinely started on statins; their QRISK2 score should be calculated

Important for me Less important

This is a patient with high risk in terms of cardiovascular disease due to the fact that he is male, has obesity, is a current smoker and has a strong family history of heart disease. Therefore it is very important to manage his risk factors. In patients with type 2 diabetes, risk should be assessed using the QRISK2 method. This formula uses cardiovascular risk factors and estimates risk of cardiovascular ischaemia within the next 10 years. If you were to assess this patient, his risk would be far greater than 25%. NICE guidelines advises that if the risk is greater than 10% then Atorvastatin 20mg at night should be started, and increased if the cholesterol does not fall. Starting 80mg of Atorvastatin would be appropriate in secondary prevention, such as following a myocardial infarction. NICE does not advise starting aspirin or clopidogrel as primary prevention unless there is a high risk for an ischaemic event, as determined by QRISK scoring, and the patient is hypertensive or being treated for hypertension. This is because the risk of gastrointestinal bleeding is thought to outweigh the benefit of reduction in cardiovascular events in such patients.

👍 👎 Discuss (3) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake

- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

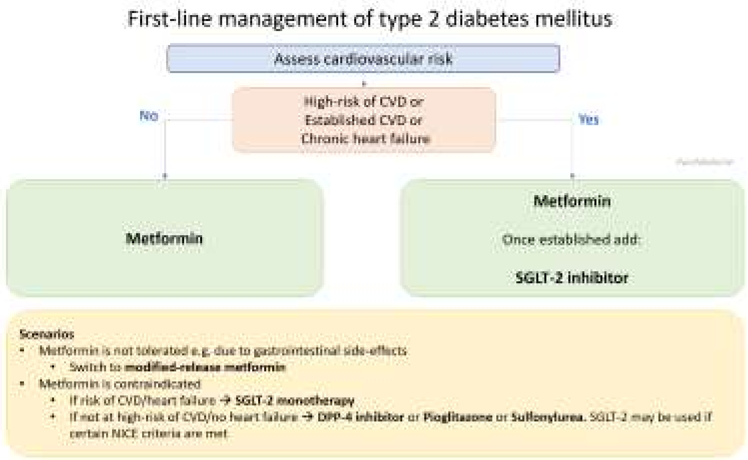
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

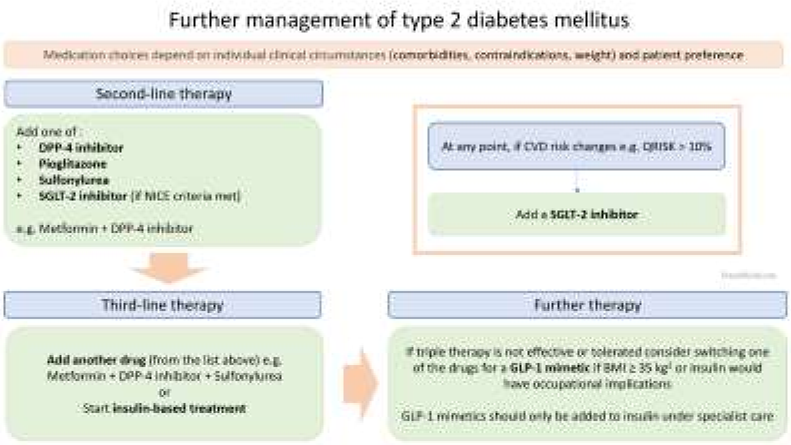
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure

- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

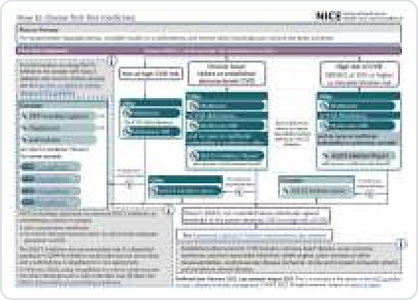
GLP-1 mimetics should only be added to insulin under specialist care

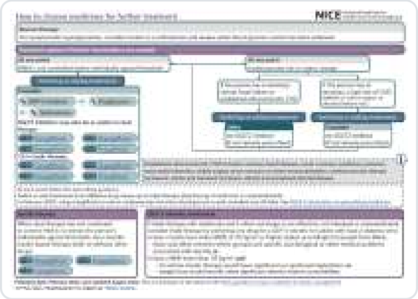
Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to

need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

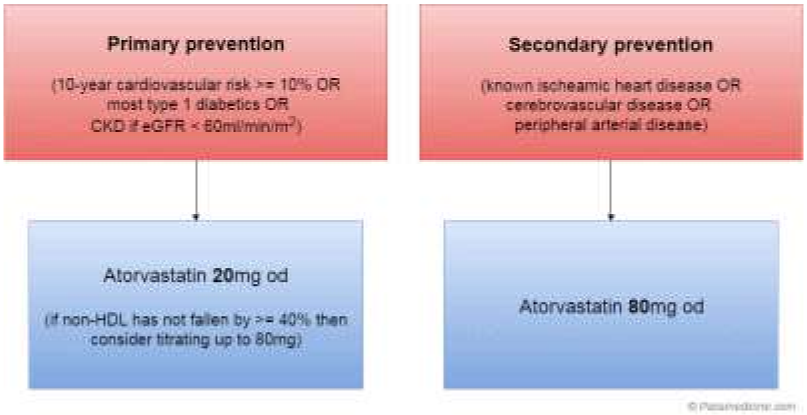
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 33 40

2014 Lipid modification guidelines

SIGN 36 41

2010 Management of diabetes

NICE 👍 56 👎 48

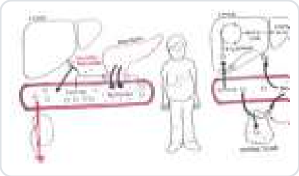
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

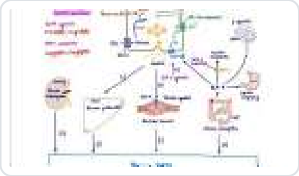
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **55.6%**

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A 32-year-old man is reviewed in the ischaemic heart disease clinic having suffered an inferior myocardial infarction. He has been diagnosed with heterozygous familial hypercholesterolaemia and started on 80mg per day of atorvastatin. His LDL cholesterol is still 3.5 mmol/l.

Which of the following is the most appropriate next intervention?

- ○ Add cholestyramine ✕
- ○ Add evolocumab ✕
- ○ Add fenofibrate ✕
- ○ Add nicotinic acid ✕
- ○ Change atorvastatin to rosuvastatin ✕

Submit answer

Reference ranges

Score: **55.6%**

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A 32-year-old man is reviewed in the ischaemic heart disease clinic having suffered an inferior myocardial infarction. He has been diagnosed with heterozygous familial hypercholesterolaemia and started on 80mg per day of atorvastatin. His LDL cholesterol is still 3.5 mmol/l.

Which of the following is the most appropriate next intervention?

	Add cholestyramine	5%
🟩	Add evolocumab	38%
	Add fenofibrate	38%
	Add nicotinic acid	11%
🟥	Change atorvastatin to rosuvastatin	8%

Although the 80mg of atorvastatin has clearly brought the LDL cholesterol much closer to target, but LDL of 3.5 mmol/l is still suboptimal given the history of an inferior myocardial infarction. Evolocumab, a PCSK9 inhibitor which interferes with degradation of the LDL receptor is the most appropriate intervention and can reduce LDL by a further 50%. Given his relatively young age and high lifetime risk of cardiovascular disease, he is exactly the type of patient likely to benefit from PCSK9 inhibition.

Cholestyramine was formerly used in the treatment of hypercholesterolaemia, it is a cholesterol binding resin, but is less effective in lowering cholesterol than a statin. Both fenofibrate and nicotinic acid have most effect in lowering triglyerides, and changing atorvastatin to rosuvastatin is likely to have limited benefit in further lowering LDL.

https://www.medicines.org.uk/emc/medicine/30627

👍 👎 Discuss (15) Improve

Next question

Familial hypercholesterolaemia

Familial hypercholesterolaemia (FH) is an autosomal dominant condition that is thought to affect around 1 in 500 people. It results in high levels of LDL-cholesterol which, if untreated, may cause early cardiovascular disease (CVD). FH is caused by mutations in the gene which encodes the LDL-receptor protein.

Case finding:

- NICE suggest that we should suspect FH as a possible diagnosis in adults with:
    - a total cholesterol level greater than 7.5 mmol/l and/or
    - a personal or family history of premature coronary heart disease (an event before 60 years in an index individual or first-degree relative)
- children of affected parents:
    - if one parent is affected by familial hypercholesterolaemia, arrange testing in children by age 10
    - if both parents are affected by familial hypercholesterolaemia, arrange testing in children by age 5

Clinical diagnosis is now based on the **Simon Broome criteria**:

- in adults total cholesterol (TC) > 7.5 mmol/l and LDL-C > 4.9 mmol/l or children TC > 6.7 mmol/l and LDL-C > 4.0 mmol/l, plus:
- for definite FH: tendon xanthoma in patients or 1st or 2nd degree relatives or DNA-based evidence of FH
- for possible FH: family history of myocardial infarction below age 50 years in 2nd degree relative, below age 60 in 1st degree relative, or a family history of raised cholesterol levels

Management

- the use of CVD risk estimation using standard tables is not appropriate in FH as they do not accurately reflect the risk of CVD
- referral to a specialist lipid clinic is usually required
- high-dose statins are usually used first-line
- first-degree relatives have a 50% chance of having the disorder and should therefore be offered screening. This includes children who should be screened by the age of 10 years if there is one affected parent
- statins should be discontinued in women 3 months before conception due to the risk of congenital defects

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The FH Foundation 👍 5 👎 4

High cholesterol during preganancy

The FH Foundation 👍 7 👎 5

Simon Broom Criteria

Public Library of Science (PLoS) 👍 5 👎 3

Cascade Screening for Familial Hypercholesterolemia (FH)

NICE 👍 4 👎 2

2008 Familial hypercholesterolaemia guidelines

Suggest link Report broken link

Media

FAMILIAL
HYPERCHOLESTEROLEMIA
Osmosis.org

Familial hypercholesterolaemia

Osmosis - YouTube 👍 10 👎 1

Report broken media

Score: **55%**

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Question 101 of 342

A 48-year-old woman is referred to the endocrinology clinic with a 6-week history of weight gain and fatigue.

Her observations are as follows:

- Temperature 36.5ºC
- Heart rate 77 beats/min
- Blood pressure 171/97mmHg
- Respiratory rate 16 breaths/min
- Oxygen saturations 98% on air

Laboratory tests:

Hb	136 g/L	(115 - 160)
Platelets	288 * 10^9/L	(150 - 400)
WBC	9.4 * 10^9/L	(4.0 - 11.0)
Na^+	142 mmol/L	(135 - 145)
K^+	3.4 mmol/L	(3.5 - 5.0)
Urea	4.1 mmol/L	(2.0 - 7.0)
Creatinine	112 µmol/L	(55 - 120)
Glucose 16 mmol/L	(4 - 7)	

Low-dose dexamethasone suppression test:

Cortisol	250 nmol/L	(119 - 618)

What is the next best investigation?

○ High-dose dexamethasone suppression test ✕

○ Insulin tolerance test ✕

○ Petrosal sinus ACTH sampling ×

○ Pituitary MRI ×

○ Urinary cortisol ×

Submit answer

Reference ranges

Score: **55%**

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Question 101 of 342 ✓

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Creatinine	112 µmol/L	(55 - 120)
Glucose 16 mmol/L	(4 - 7)	

Low-dose dexamethasone suppression test:

Cortisol	250 nmol/L	(119 - 618)

What is the next best investigation?

High-dose dexamethasone suppression test	64%
Insulin tolerance test	12%

Petrosal sinus ACTH sampling	6%
Pituitary MRI	5%
Urinary cortisol	12%

The high-dose dexamethasone suppression test is useful for distinguishing between ACTH dependent (e.g. pituitary source) and non-ACTH dependent (e.g. ectopic and adrenal source) causes of Cushings's syndrome

Important for me Less important

This patient has Cushing's syndrome. Due to increased glucocorticoid production, symptoms include weight gain and hyperglycaemia as seen in this patient. Hypertension is another common finding also seen here. Laboratory tests may reveal hypokalaemic metabolic acidosis. The best test to diagnose Cushing's syndrome is the low-dose dexamethasone test. This has been done for this patient and shows a cortisol level that has not been suppressed despite exogenous steroid use. Once Cushing's syndrome has been confirmed, additional localisation tests need to be completed. One of these includes the high-dose dexamethasone suppression test which is used to discriminate between Cushing's disease (i.e. pituitary adenoma) and ectopic ACTH production. Suppressed cortisol levels following high doses of glucocorticoids confirms a pituitary cause, as opposed to normal levels of cortisol that suggest an adrenal cause.

The insulin stress test is used to differentiate Cushing's syndrome from pseudo-Cushing's. Pseudo-Cushing's presents similarly to Cushing's syndrome; however, rather than being caused by excessive corticosteroid levels, it is due to alcohol excess or severe depression. There is nothing in this patient's history to suggest depression or alcohol excess. Furthermore, the positive finding following the low-dose dexamethasone suppression test points away from a diagnosis of pseudo-Cushing's.

Petrosal sinus ACTH sampling is another investigation that can be used to help distinguish between a pituitary or adrenal cause of Cushing's syndrome. However, it is an invasive investigation and easier tests such as the high-dose dexamethasone suppression test should be considered first.

A pituitary MRI can be used to diagnose a pituitary cause of Cushing's syndrome. However, it is not the next recommended investigation. Rather, a high-dose dexamethasone suppression test is easier and cheaper to complete and should be completed next.

Urinary cortisol is used as an initial investigation in the diagnosis of Cushing's syndrome. It is not a specific localisation test.

👍 👎 💬 Discuss (10) Improve

Next question

Cushing's syndrome: investigations ★

This section will consider:

- the general lab findings consistent with Cushing's syndrome
- tests to confirm whether a patient indeed has Cushing's syndrome
- tests to find the underlying cause of Cushing's syndrome

It is useful to bear in mind the possible causes:

- iatrogenic: corticosteroid therapy
- ACTH-dependent causes
  - Cushing's disease (a pituitary adenoma → ACTH secretion)
  - ectopic ACTH secretion secondary to a malignancy
- ACTH-independent causes
  - adrenal adenoma

General findings consistent with Cushing's syndrome

A hypokalaemic metabolic alkalosis may be seen, along with impaired glucose tolerance.

Ectopic ACTH secretion (e.g. secondary to small cell lung cancer) is characteristically associated with very low potassium levels.

Tests to confirm Cushing's syndrome

The three most commonly used tests are:

- overnight (low-dose) dexamethasone suppression test
  - this is the most sensitive test and is now used first-line to test for Cushing's syndrome
  - patients with Cushing's syndrome do not have their morning cortisol spike suppressed
- 24 hr urinary free cortisol
  - two measurements are required
- bedtime salivary cortisol
  - two measurements are required

Localisation tests

The first-line localisation is 9am and midnight plasma ACTH (and cortisol) levels. If ACTH is suppressed then a non-ACTH dependent cause is likely such as an adrenal adenoma

High-dose dexamethasone suppression test

The high-dose dexamethasone suppression test may be used to localise the pathology resulting in Cushing's syndrome. This test may be interpreted as follows:

Cortisol	ACTH	Interpretation
Not suppressed	Suppressed	Cushing's syndrome due to other causes (e.g. adrenal adenomas)
Suppressed	Suppressed	Cushing's disease (i.e. pituitary adenoma → ACTH secretion)
Not suppressed	Not suppressed	Ectopic ACTH syndrome

Other tests

CRH stimulation

- if pituitary source then cortisol rises
- if ectopic/adrenal then no change in cortisol

Petrosal sinus sampling of ACTH may be needed to differentiate between pituitary and ectopic ACTH secretion.

An insulin stress test is used to differentiate between true Cushing's and pseudo-Cushing's.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypothalamus
Anterior Pituitary
Adrenal Gland
Kidney
Dexamethasone 1mg
ZERO TO FINALS

Understanding The Dexamethasone Suppression Test

Zero to Finals - YouTube 👍 31 👎 3

CUSHING SYNDROME

Cushing syndrome

Osmosis - YouTube 👍 7 👎 1

CUSHING'S SYNDROME

Understanding Cushing's Syndrome

Zero To Finals - YouTube 👍 1 👎 1

Report broken media

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Question 102 of 342

A 56-year-old woman presents to endocrinology clinic. She has been referred after CT scanning revealed a 3mm mass in the thyroid. She originally had a CT scan of her neck as she has rheumatoid arthritis and neck pain, and was due to undergo general anesthesia and intubation, and the anesthetist wanted to ensure that her neck was stable. She was due to have a coronary arterial bypass graft. She has a past medical history of type 2 diabetes mellitus, high cholesterol and hypertension. She has no symptoms of overactive or underactive thyroid function. What is the most appropriate action?

- ○ Reassure and discharge back to her GP ×
- ○ Arrange for US guided FNAC ×
- ○ Arrange for nuclear scan ×
- ○ Repeat CT in 6 months ×
- ○ Refer for urgent hemithyroidectomy ×

Submit answer

Reference ranges

Score: **55.4%**

1 ✓
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3 ✓

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A 56-year-old woman presents to endocrinology clinic. She has been referred after CT scanning revealed a 3mm mass in the thyroid. She originally had a CT scan of her neck as she has rheumatoid arthritis and neck pain, and was due to undergo general anesthesia and intubation, and the anesthetist wanted to ensure that her neck was stable. She was due to have a coronary arterial bypass graft. She has a past medical history of type 2 diabetes mellitus, high cholesterol and hypertension. She has no symptoms of overactive or underactive thyroid function. What is the most appropriate action?

🟩	Reassure and discharge back to her GP	21%
🟥	Arrange for US guided FNAC	53%
	Arrange for nuclear scan	18%
	Repeat CT in 6 months	7%
	Refer for urgent hemithyroidectomy	1%

A non-palpable thyroid mass less than 1cm in size found incidentally without worrying features does not need biopsy

Important for me Less important

This thyroid mass is not concerning. A patient with a non-palpable nodule which has been found on imaging and is less than 1cm in size does not need biopsy and can be safely managed in primary care. However, if the patient did have a worrying features such as age outside of 20-60 years old, had rapid growth of mass, vocal cord paralysis or had a history of neck irradiation or thyroid cancer then urgent investigations should be done, primarily a US guided FNAC. Repeating the CT would be inappropriate in terms of the amount of radiation exposure. A diagnostic hemithyroidectomy would be appropriate following a biopsy showing a Thy3a, Thy3f or a Thy4 lesion.

👍 👎 Discuss (13) Improve

Next question

Thyroid disorders: a very basic introduction ★

Disorders of thyroid function are very commonly encountered in clinical practice. Around 2% of the UK population has hypothyroidism (an under active thyroid gland) whilst around 1% have thyrotoxicosis (an over active gland). Both hypothyroidism and hyperthyrodism (also known as thyrotoxicosis) are around 10 times more common in women than men.

Structure and function

The thyroid gland is one of the largest endocrine organs in the body. It is a bi-lobed structure which is found in the anterior neck. As with many endocrine organs, it is part of a hypothalamus-pituitary-end organ system with negative feedback cycles to maintain normal circulating levels of the hormone, in this case thyroxine and triiodothyronine.

On a simple level the hypothalamus secretes thyrotropin-releasing hormone (TRH) which stimulates the anterior pituitary to secrete thyroid-stimulating hormone (TSH). This then acts on the thyroid gland increasing the production of thyroxine (T4) and triiodothyronine (T3), the two main thyroid hormones. These then act on a wide variety of tissues, helping to regulate the use of energy sources, protein synthesis, and controls the body's sensitivity to other hormones.

How are thyroid problems classified?

Hypothyroidism may be classified as follows:

- primary hypothyroidism: there is a problem with the thyroid gland itself, for example an autoimmune disorder affecting thyroid tissue (see below)
- secondary hypothyroidism: usually due to a disorder with the pituitary gland (e.g.pituitary apoplexy) or a lesion compressing the pituitary gland
- congenital hypothyroidism: due to a problem with thyroid dysgenesis or thyroid dyshormonogenesis

Whilst there are a number of causes thyrotoxicosis the vast majority are primary in nature. Congenital thyrotoxicosis is not seen and secondary hyperthyroidism is rare, account for less than 1% of cases.

What causes thyroid problems?

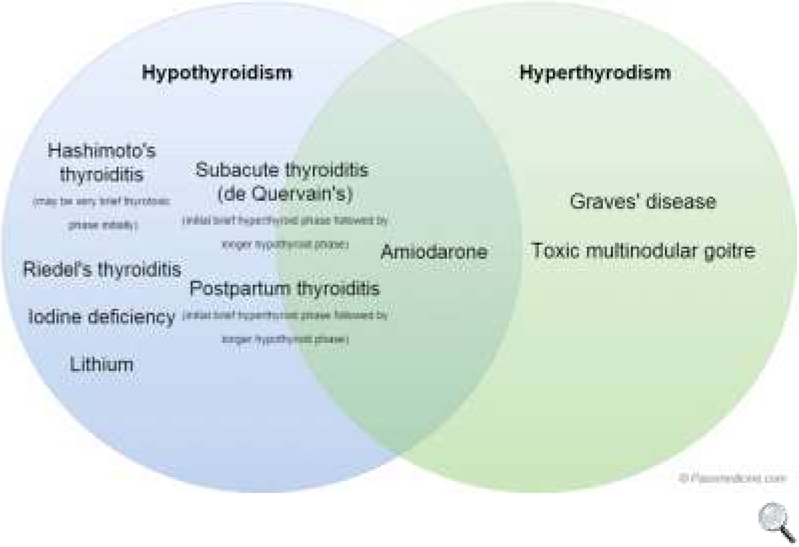
The majority of thyroid problems seen in the developed world are a consequence of autoimmunity.

The table below shows the different autoimmune problems which cause thyroid dysfunction:

	Hypothyroidism	**Thyrotoxicosis**
Most common cause	Hashimoto's thyroiditis • most common cause in the developed world • autoimmune disease, associated with type 1 diabetes mellitus, Addison's or pernicious anaemia • may cause transient thyrotoxicosis in the acute phase • 5-10 times more common in women	Graves' disease • most common cause of thyrotoxicosis • as well as typically features of thyrotoxicosis other features may be seen including thyroid eye disease

	Hypothyroidism	**Thyrotoxicosis**
Other causes	Subacute thyroiditis (de Quervain's) • associated with a painful goitre and raised ESR Riedel thyroiditis • fibrous tissue replacing the normal thyroid parenchyma • causes a painless goitre Postpartum thyroiditis Drugs • lithium • amiodarone Iodine deficiency • the most common cause of hypothyroidism in the developing world	Toxic multinodular goitre • autonomously functioning thyroid nodules that secrete excess thyroid hormones Drugs • amiodarone

It should be remembered that a lot of the conditions mentioned above don't always cause either hypothyroidism or hyperthyroidism, there is sometimes some overlap, as shown below:


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Riedel's thyroiditis
Iodine deficiency
Lithium
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Symptoms and signs

Thyroid disorders can present in a large variety of ways. Often (but not always) the symptoms present are the opposite depending on whether the thyroid gland is under or over active, for example hypothyroidism may result in weight gain whilst thyrotoxicosis normally leads to weight loss

Feature	Hypothyroidism	Thyrotoxicosis
General	Weight gain **Lethargy** Cold intolerance	Weight loss 'Manic', restlessness Heat intolerance
Cardiac	-	**Palpitations**, may even provoke arrhythmias e.g. atrial fibrillation
Skin	Dry (anhydrosis), cold, yellowish skin Non-pitting oedema	Increased sweating Pretibial myxoedema: erythematous,

Feature	Hypothyroidism	Thyrotoxicosis
	(e.g. hands, face) Dry, coarse scalp hair, loss of lateral aspect of eyebrows	oedematous lesions above the lateral malleoli Thyroid acropachy: clubbing
Gastrointestinal	Constipation	Diarrhoea
Gynaecological	Menorrhagia	Oligomenorrhea
Neurological	Decreased deep tendon reflexes Carpal tunnel syndrome	**Anxiety** **Tremor**

Investigations and diagnosis

The principle investigation is 'thyroid function tests', or TFTs for short:

- these primarily look at serum TSH and T4 levels
- T3 can be measured but is only useful clinically in a small number of cases
- remember that TSH and T4 levels will often be 'opposite' in cases of primary hypo- or hyperthyroidism. For example in hypothyroidism the T4 level is low (i.e. not enough thyroxine) but the TSH level is high, because the hypothalamus/pituitary has detected low levels of T4 and is trying to get the thyroid gland to produce more
- TSH levels are more sensitive than T4 levels for monitoring patients with existing thyroid problems and are often used to guide treatment

The table below shows how thyroid function tests are interpreted:

Diagnosis	TSH	Free T4	Notes
Thyrotoxicosis (e.g. Graves' disease)	Low	High	
Primary hypothyroidism (e.g. Hashimoto's thyroiditis)	High	Low	
Secondary hypothyroidism	Low	Low	
Sick euthyroid syndrome	Low	Low	Common in hospital inpatients. Changes are reversible upon recovery from the systemic illness and no treatment is usually needed
Subclinical hypothyroidism	High	Normal	This is a common finding and represents patients who are 'on the way' to developing hypothyroidism but still have normal thyroxine levels. Note how the TSH levels, as mentioned above, are a more sensitive and early marker of thyroid problems
Poor compliance with thyroxine	High	Normal	Patients who are poorly compliant may only take their thyroxine in the days before a routine blood test. The thyroxine levels are hence normal but the TSH 'lags' and reflects longer term low thyroxine levels

A number of thyroid autoantibodies can be tested for (remember the majority of thyroid disorders are autoimmune). The 3 main types are:

- Anti-thyroid peroxidase (anti-TPO) antibodies
- TSH receptor antibodies
- Thyroglobulin antibodies

There is significant overlap between the type of antibodies present and particular diseases, but generally speaking TSH receptor antibodies are present in around 90-100% of patients with Graves' disease and anti-TPO antibodies are seen in around 90% of patients with Hashimoto's thyroiditis.

Other tests include:

- nuclear scintigraphy; toxic multinodular goitre reveals patchy uptake

Treatment

This clearly depends on the cause. For patients with hypothyrodism thyroxine is given in the form of levothyroxine to replace the underlying deficiency.

Patients with thyrotoxicosis may be treated with:

- propranolol: this is often used at the time of diagnosis to control thyrotoxic symptoms such as tremor
- carbimazole: blocks thyroid peroxidase from coupling and iodinating the tyrosine residues on thyroglobulin → reducing thyroid hormone production. Agranulocytosis is an important adverse effect to be aware of
- radioiodine treatment

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 7 👎 5

2017 Thyrotoxicosis â€“ investigation and management

Suggest link

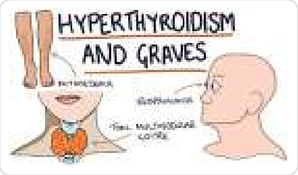
Report broken link

Media

HYPOTHYROIDISM
CAUSES

Hypothyroidism and Hashimoto's Thyroiditis: Visual Explanation for Students

Zero To Finals - YouTube 👍 2 👎 0


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube

👍 0 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

[Hyperthyroidism Overview (causes, pathophysiology)]

Armando Hasudungan - YouTube 👍 0 👎 0

THYROID
FUNCTION
TESTS

Understanding Thyroid Function Tests

Zero To Finals - YouTube 👍 0 👎 0

Report broken media

Score: **54.9%**

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Back to top

A 28-year-old woman has presented with a 5 month history of weight loss (despite an increase in appetite), tremor, loose bowels, and heat intolerance. She has otherwise been well and her only significant family history is that her brother has alopecia areata. She tells you that she had a positive pregnancy test last week and is awaiting her booking appointment. On examination, she appears anxious and her heart rate is 105 beats/minute. She has a tremor when her arms are outstretched and her eyes appear large. She also has a goitre. The rest of her examination is unremarkable. Her blood results find hyperthyroidism. Which of the following medications are most suited to treat her hyperthyroidism?

- ○ Propylthiouracil ×
- ○ Carbimazole ×
- ○ Radioactive iodine ×
- ○ Carbimazole and Levothyroxine ×
- ○ Levothyroxine ×

Submit answer

Reference ranges

Score: **54.9%**

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← Question 103 of 342 ✓ →

A 28-year-old woman has presented with a 5 month history of weight loss (despite an increase in appetite), tremor, loose bowels, and heat intolerance. She has otherwise been well and her only significant family history is that her brother has alopecia areata. She tells you that she had a positive pregnancy test last week and is awaiting her booking appointment. On examination, she appears anxious and her heart rate is 105 beats/minute. She has a tremor when her arms are outstretched and her eyes appear large. She also has a goitre. The rest of her examination is unremarkable. Her blood results find hyperthyroidism. Which of the following medications are most suited to treat her hyperthyroidism?

Propylthiouracil	86%
Carbimazole	11%
Radioactive iodine	1%
Carbimazole and Levothyroxine	2%
Levothyroxine	0%

During early pregnancy, propylthiouracil should be used. The block and replace strategy is not advised as it can lead to problems in the fetus and radioactive iodine is contraindicated. Please see the link below for more information: https:www.evidence.nhs.uk/formulary/bnf/current/6-endocrine-system/62-thyroid-and-antithyroid-drugs/622-antithyroid-drugs

Discuss (1) | Improve

Next question

Pregnancy: thyroid problems

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum

- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link Report broken link

Score: **55.3%**

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A 29-year-old female is admitted to the Emergency Department following an episode of collapse. She denies prodromal symptoms and woke up after an undetermined period to find herself lying face down on the ground. She has recently consulted her GP regarding feelings of generalised weakness, intermittent palpitations and dizziness.

Her past medical history is remarkable for hypothyroidism and rheumatoid arthritis, previously managed with infliximab, and complicated 3 months ago by a diagnosis of TB. Her regular medications include methotrexate, folic acid, levothyroxine, artificial tears, rifampicin, and isoniazid.

During the examination, the patient complains of recurrent palpitations. The cardiac monitor shows broad-complex tachycardia. Her blood pressure is stable at 117/68mmHg and she is given a bolus of amiodarone.

Her venous blood gas is as follows:

pH	7.31
pCO2	4.3 kPa
pO2	6.3 kPa
Na+	137 mmol/l
K+	2.1 mmol/l
Cl-	114 mmol/l
iCa2+	1.05 mmol/l
Glucose	5.4 mmol/l
HCO3	15.6 mmol/l
BE	-9.5 mmol/l

Dr. Jamil Alrajhi

Some additional investigations are requested:

Urine dipstick	pH 5.0 glucose ++ protein +
XR chest	unremarkable
XR abdomen	normal bowel gas pattern, no evidence of abnormal renal calcification

What is the most likely cause of the metabolic abnormalities described?

- ○ Addison's disease ×
- ○ Fanconi syndrome ×
- ○ Rheumatoid arthritis ×
- ○ Sjogren's syndrome ×
- ○ Liver cirrhosis ×

Submit answer

Reference ranges ⌄

Score: **55.3%**

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Na+	137 mmol/l
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Cl-	114 mmol/l
iCa2+	1.05 mmol/l
Glucose	5.4 mmol/l
HCO3	15.6 mmol/l
BE	-9.5 mmol/l

Some additional investigations are requested:

Urine dipstick	pH 5.0 glucose ++ protein +
XR chest	unremarkable
XR abdomen	normal bowel gas pattern, no evidence of abnormal renal calcification

What is the most likely cause of the metabolic abnormalities described?

	Addison's disease	11%
🟩	Fanconi syndrome	68%
	Rheumatoid arthritis	10%
🟥	Sjogren's syndrome	10%
	Liver cirrhosis	1%

The patient has a normal anion gap hyperchloraemic metabolic acidosis associated with hypokalaemia. This, in combination with the absence of GI symptoms, should alert you to the possibility of underlying renal tubular acidosis.

Fanconi syndrome is a disorder of proximal renal tubular function that leads to abnormal loss of bicarbonate, glucose, potassium, phosphate, uric acid and amino acids in the urine. Patients have features of type 2 renal tubular acidosis, accompanied by hypophosphataemia, glycosuria, and aminoaciduria. Presenting features include polyuria, polydipsia, osteomalacia (rickets and growth failure in children) and symptoms secondary to the electrolyte abnormalities associated with the disorder. Rifampicin has recently been implicated in the development of Fanconi syndrome and is the most likely cause in this case.

Rheumatoid arthritis and Sjogren's syndrome are causes of type 1 (distal) renal tubular acidosis; a failure of hydrogen ion secretion in the distal tubule that causes hypokalaemia and nephrocalcinosis. The absence of abnormal renal calcification on the patient's x-ray points away from this as the diagnosis.

Addison's disease typically causes hyperkalaemia, often associated with other electrolyte abnormalities including hyponatraemia and hypoglycaemia. It would, therefore, be inconsistent with the biochemistry provided.

Liver cirrhosis does not typically cause renal tubular acidosis unless it is itself a manifestation of Wilson's disease. There is nothing in the question to suggest Wilson's disease as the underlying diagnosis, however, and the occurrence of this pattern of metabolic abnormality would still be predicated on the development of a complicating Fanconi syndrome.

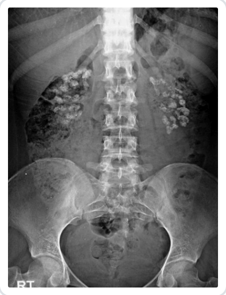
👍 👎 Discuss (5) Improve

Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)
- decreased HCO3- reabsorption in proximal tubule

- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

AI + Q 123

Next question

B *I* A

Textbooks

High-yield textbook

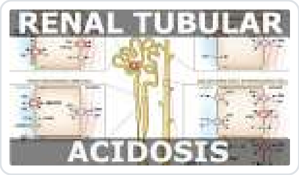
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

[Renal tubular acidosis]

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
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[Renal tubular acidosis]

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Question 105 of 342

A 17-year-old male is referred to the endocrinology clinic due to a lack of secondary sex characteristics and low serum testosterone. He has never completely developed pubertal body hair or muscle mass, and has small testicles. His past medical history includes a cleft palate at birth. He also finds it difficult to smell things. What is the most likely underlying diagnosis?

- ○ Klinefelter's syndrome ×
- ○ Androgen insensitivity syndrome ×
- ○ Pituitary adenoma ×
- ○ Testicular tumour ×
- ○ Kallman syndrome ×

Submit answer

Reference ranges

Score: **54.8%**

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Question 105 of 342 ✓

A 17-year-old male is referred to the endocrinology clinic due to a lack of secondary sex characteristics and low serum testosterone. He has never completely developed pubertal body hair or muscle mass, and has small testicles. His past medical history includes a cleft palate at birth. He also finds it difficult to smell things. What is the most likely underlying diagnosis?

Klinefelter's syndrome	9%
Androgen insensitivity syndrome	2%
Pituitary adenoma	1%
Testicular tumour	0%
Kallman syndrome	88%

Anosmia in the context of male hypogonadism, low testosterone and normal or low FSH and LH makes Kallman Syndrome likely

Important for me Less important

The correct answer is Kallman syndrome, largely indicated from the history of primary hypogonadism in a young patient lacking secondary sex characteristics and anosmia. The history of cleft palate also is suggestive and helps differentiate from Klinefelter's syndrome. Androgen insensitivity syndrome would present as phenotypically female but genetically male.

Discuss (3) Improve

Next question

Kallmann's syndrome

Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. It is usually inherited as an X-linked recessive trait. Kallmann's syndrome is thought to be caused by failure of GnRH-secreting neurons to migrate to the hypothalamus.

The clue given in many questions is lack of smell (anosmia) in a boy with delayed puberty.

Features

- 'delayed puberty'
- hypogonadism, cryptorchidism
- anosmia
- sex hormone levels are low
- LH, FSH levels are inappropriately low/normal
- patients are typically of normal or above-average height

Cleft lip/palate and visual/hearing defects are also seen in some patients

Management

- testosterone supplementation
- gonadotrophin supplementation may result in sperm production if fertility is desired later in life

+ Q 123

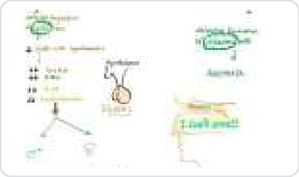
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Kallmann Syndrome mnemonic

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KALLMANN
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Kallman's syndrome

Osmosis - YouTube 👍 8 👎 2

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Question 106 of 342

A 37-year-old man presents to the endocrinology clinic for annual review of type 1 diabetes mellitus. He was diagnosed with diabetes following an admission with diabetic ketoacidosis (DKA) at the age of 21. Since then he has been managed with a basal-bolus regime of insulin. He has since then had one admission with DKA in the year following his diagnosis due to excessive alcohol intake and has since then significantly reduced his alcohol intake. He had retinal photography 11 months ago which he reports as normal. His blood pressure is 135/81mmHg. He had blood tests showing a normal cholesterol as well as HbA1c at 42mmol/mol. He has given urine today to measure the albumin to creatinine ratio, but there was no protein in the sample on dipstick testing. What is the most appropriate change to make his medications?

○ Increase long-acting insulin dose ×

○ Start ACE inhibitor ×

○ Start calcium-channel blocker ×

○ Start statin ×

○ Decrease long-acting insulin dose ×

Submit answer

Reference ranges

Score: **55.2%**

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105 ✓

106 -

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← Question 106 of 342 ✓ →

A 37-year-old man presents to the endocrinology clinic for annual review of type 1 diabetes mellitus. He was diagnosed with diabetes following an admission with diabetic ketoacidosis (DKA) at the age of 21. Since then he has been managed with a basal-bolus regime of insulin. He has since then had one admission with DKA in the year following his diagnosis due to excessive alcohol intake and has since then significantly reduced his alcohol intake. He had retinal photography 11 months ago which he reports as normal. His blood pressure is 135/81mmHg. He had blood tests showing a normal cholesterol as well as HbA1c at 42mmol/mol. He has given urine today to measure the albumin to creatinine ratio, but there was no protein in the sample on dipstick testing. What is the most appropriate change to make his medications?

Increase long-acting insulin dose	4%
Start ACE inhibitor	37%
Start calcium-channel blocker	2%
Start statin	46%
Decrease long-acting insulin dose	12%

Patients with T1DM for over 10 years should be considered for statin therapy atorvastatin 20mg OD

Important for me Less important

This is a patient having a diabetic review. He has had diabetes for 16 years and this increases his cardiovascular risk and NICE guidelines advise that all patients with type 1 diabetes mellitus who have had their diagnosis for over 10 years should be offered a statin, even if cholesterol is not measured to be high. His HbA1c demonstrates that he has good control of his diabetes; if it had been higher then increasing long-acting dose of insulin could be considered, but even then the capillary glucose levels should be reviewed through the day to see if it would be more appropriate to increase the bolus doses instead. As there is not evidence of episodes of hypoglycaemia the dose of insulin should not be decreased. Patient should aim to maintain a blood pressure <140/80mmHg if no end-organ damage, and <130/80mmHg of there is end organ damage, such as retinopathy, neuropathy or nephropathy. There is no evidence that the patient has any of these complications. ACE inhibitors should be started if there is any urinary protein, but so far this is negative.

👍 👎 Discuss (3) Improve

Next question

Diabetes mellitus: management of type 1 ★

The long-term management of type 1 diabetics is an important and complex process requiring the input of many different clinical specialties and members of the healthcare team. A diagnosis of type 1 diabetes can still reduce the life expectancy of patients by 13 years and the micro and macrovascular complications are well documented.

NICE released guidelines on the diagnosis and management of type 1 diabetes in 2015. We've only highlighted a very select amount of the guidance here which will be useful for any clinician looking after a patient with type 1 diabetes.

HbA1c
- should be monitored every 3-6 months
- adults should have a target of HbA1c level of 48 mmol/mol (6.5%) or lower. NICE do however recommend taking into account factors such as the person's daily activities, aspirations, likelihood of complications, comorbidities, occupation and history of hypoglycaemia

Self-monitoring of blood glucose

- recommend testing at least 4 times a day, including before each meal and before bed
- more frequent monitoring is recommended if frequency of hypoglycaemic episodes increases; during periods of illness; before, during and after sport; when planning pregnancy, during pregnancy and while breastfeeding

Blood glucose targets

- 5-7 mmol/l on waking and
- 4-7 mmol/l before meals at other times of the day

Type of insulin

- offer multiple daily injection basal-bolus insulin regimens, rather than twice-daily mixed insulin regimens, as the insulin injection regimen of choice for all adults
- twice-daily insulin detemir is the regime of choice. Once-daily insulin glargine or insulin detemir is an alternative
- offer rapid-acting insulin analogues injected before meals, rather than rapid-acting soluble human or animal insulins, for mealtime insulin replacement for adults with type 1 diabetes

Metformin

- NICE recommend considering adding metformin if the BMI >= 25 kg/m²

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 8 👎 6

2015 Diabetes (type 1 and type 2) in children and young people

NICE 👍 15 👎 7

2015 Type 1 diabetes in adults: diagnosis and management

Suggest link

Report broken link

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A 55-year-old woman attends the emergency department with abdominal pain. She complains of right-sided loin pain which radiates to her groin. She is otherwise well. She has a past medical history of Sjogren's syndrome. On examination, you note right-sided renal angle tenderness.

Blood results are as follows:

Hb	126 g/L	Male: (135-180) Female: (115 - 160)
Platelets	245 * 10^9/L	(150 - 400)
WBC	6.2 * 10^9/L	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	2.5 mmol/L	(3.5 - 5.0)
Cl^-	120 mmol/L	(96 - 106)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	56 µmol/L	(55 - 120)
Bicarbonate	14 mmol/L	(23 - 29)
Glucose	22.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

- ○ Addison's disease ✕
- ○ Diabetic ketoacidosis ✕
- ○ Type 1 renal tubular acidosis ✕
- ○ Type 2 renal tubular acidosis ✕

○ Type 4 renal tubular acidosis ×

Submit answer

Reference ranges

Score: **55.7%**

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Question 107 of 342 ✓

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Cl^-	120 mmol/L	(96 - 106)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	56 µmol/L	(55 - 120)
Bicarbonate	14 mmol/L	(23 - 29)
Glucose	22.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

Addison's disease	1%
Diabetic ketoacidosis	2%
🟩 Type 1 renal tubular acidosis	87%
Type 2 renal tubular acidosis	9%

Type 4 renal tubular acidosis 1%

Type 1 (**distal**) renal tubular acidosis may be caused by Sjogren's syndrome

Important for me Less important

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 2.5) - (120 + 14) = 4.5.

Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include ('ABCD'):
- Addison's
- Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
- Chloride excess
- Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:
- Lactate
- Toxins (e.g. methanol, paracetamol, propylene glycol)
- Ketones
- Renal failure

Type 1 renal tubular acidosis is correct. The presence of kidney stones, hypokalemia, NAGMA, and a past medical history of Sjogren's favour the diagnosis of type 1 RTA.

Addison's disease is incorrect. Although this can also cause a NAGMA, hyperkalemia would be expected as a consequence of hypoaldosteronism. Furthermore, patients with Addison's will often be hypoglycaemic.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia and likely has undiagnosed diabetes mellitus, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This can also present with hypokalemia and NAGMA, however, there is not an association with Sjogren's making this a less likely diagnosis.

Type 4 renal tubular acidosis is incorrect. Although diabetes is a cause of type 4 RTA, this condition would be associated with hyperkalemia in contrast to the hypokalemia demonstrated in this case.

👍 👎 Discuss (4) Improve

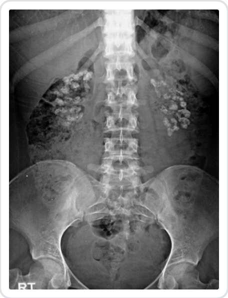
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

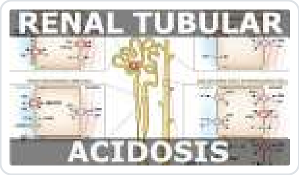
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
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Renal tubular acidosis

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Score: **56.1%**

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Question 108 of 342

A 57-year-old female presents after noticing a lump in her neck. She reports it being non-tender and she is only concerned about it for cosmetic purposes. She reports no other symptoms. Her past medical history includes hypertension and constipation. Her current medications include ramipril, irbesartan, amlodipine and furosemide. No family history is unavailable. On examination, her neck lump is hard, non-tender and measures 2cm by 1 cm, moves with swallowing but not with tongue protrusion. Her heart rate is 84 beats/min and blood pressure 213/130 mmHg. Examination of her joints was unremarkable with no excessive laxity.

Her blood tests are as follows:

Hb	112 g/l
Platelets	349 * 10^9/l
WBC	7.2 * 10^9/l
Na^+	138mmol/l
K^+	4.2 mmol/l
Urea	6.9 mmol/l
Creatinine	72 µmol/l
Adjusted calcium	3.1 mmol/l
Phosphate	0.40 mmol/l

She undergoes an outpatient ultrasound and fine needle aspiration of her neck lump. After her procedure, the recovery nurses were concerned regarding her persistent hypertension and the patient is admitted for further investigation. She develops a mild headache with no visual disturbances, resolving on its own after her blood pressure falls to 182/101 mmHg two hours later. Urinary metanephrine collection was positive.

Which investigation is likely to produce the underlying diagnosis?

- ○ Genetic testing for RET mutation ✕
- ○ Genetic testing for germline VHL mutation ✕
- ○ Genetic testing for NF2 mutation on chromosome 22 ✕
- ○ CT chest/abdomen/pelvis with contrast ✕
- ○ Urinary calcium collection ✕

Submit answer

Reference ranges ⌄

Score: **56.1%**

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Phosphate	0.40 mmol/l

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Which investigation is likely to produce the underlying diagnosis?

Genetic testing for RET mutation	87%
Genetic testing for germline VHL mutation	2%
Genetic testing for NF2 mutation on chromosome 22	3%
CT chest/abdomen/pelvis with contrast	7%
Urinary calcium collection	1%

The clinical collection of phaeochromocytoma and hyperparathyroidism should raise suspicion of multiple endocrine neoplasia type 2a, which is a genetic syndrome of near 100% penetrance caused by a mutation of the RET oncogene. The addition of a thyroid lump makes this scenario more suggestive of MEN 2, with medullary thyroid carcinoma completing the diagnosis. MEN2b is subtly differentiated from MEN2a by the lack of hyperparathyroidism and Marfanoid features. Unlikely MEN 1, genetic screening of family members for hyperparathyroidism and germline mutation is useful for long-term surveillance and monitoring. VHL and NF 2 mutations refer to von-Hippel Lindau syndrome and neurofibromatosis type 2 respectively: VHL presents classically as a combination of retinal haemangioblastomas, renal cell carcinomas and phaeochromocytomas while NF2 typically present with intracranial tumours, bilateral Schwannomas almost in all patients.

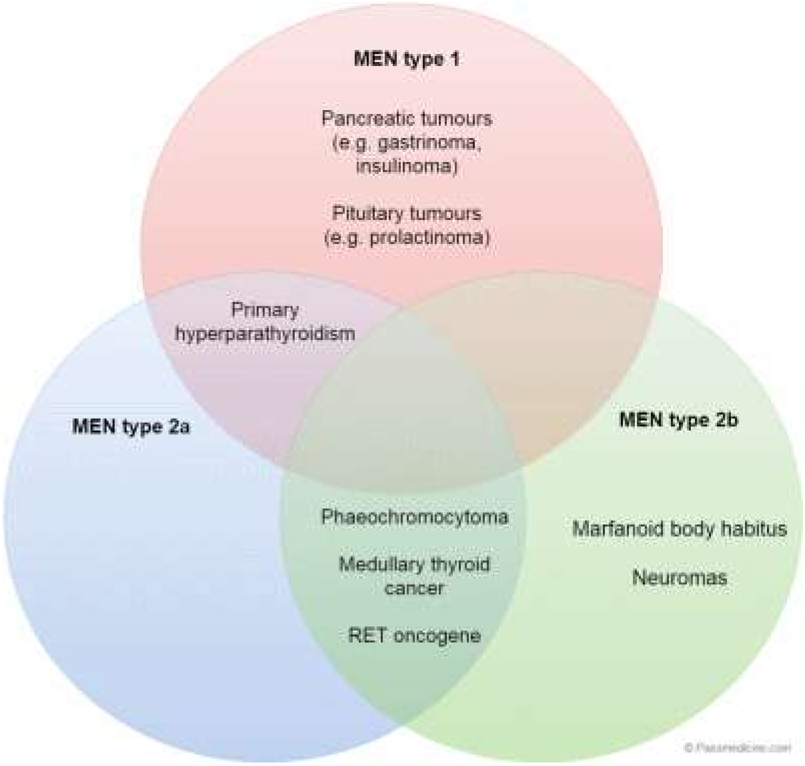
👍 👎 Discuss (3) Improve

Next question

Multiple endocrine neoplasia ★

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
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Venn diagram showing the different types of MEN and their associated features

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PANCREATIC
NEUROENDOCRINE
NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **56.5%**

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← Question 109 of 342 →

A middle age woman is being treated for symptomatic hypercalcaemia associated with a squamous cell lung cancer (serum calcium 3.60 mmol/L). She is slow to respond to initial measures of saline hydration and intravenous pamidronate. Whilst awaiting surgical resection for her underlying cancer what may be the next best step in her management?

Na^+	142 mmol/l
K^+	4.3 mmol/l
Urea	7.0 mmol/l
Creatinine	89 µmol/l
Glucose	4.8 mmol/l

What is the most appropriate management?

- ○ High dose loop diuretics ×
- ○ Calcitonin 4 units/kg ×
- ○ Insulin actrapid 50 units in 50% dextrose ×
- ○ IV colloid administration instead of crystalloid ×
- ○ Plasma exchange ×

Submit answer

Reference ranges

Score: **56.5%**

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Back to top

A middle age woman is being treated for symptomatic hypercalcaemia associated with a squamous cell lung cancer (serum calcium 3.60 mmol/L). She is slow to respond to initial measures of saline hydration and intravenous pamidronate. Whilst awaiting surgical resection for her underlying cancer what may be the next best step in her management?

Na^+	142 mmol/l
K^+	4.3 mmol/l
Urea	7.0 mmol/l
Creatinine	89 µmol/l
Glucose	4.8 mmol/l

What is the most appropriate management?

High dose loop diuretics	11%
[✓ Calcitonin 4 units/kg]	84%
Insulin actrapid 50 units in 50% dextrose	1%
IV colloid administration instead of crystalloid	2%
Plasma exchange	1%

General symptoms of hypercalcaemia may include malaise, lethargy, depression, dehydration and can lead to depressed consciousness. Bone pain and abdominal pain may feature and can be summarised by the classic 'bones, stones, moans and abdominal groans'.

Alongside searching for the underlying cause, management initially involves aggressive rehydration, typically 4-6 L saline on the first day. Bisphosphonates act by interfering with osteoclastic bone resorption and typically IV pamidronate is used at a dose of 60-90mg over 2-4 hours. Calcitonin (extracted from salmon) also interferes with osteoclast activity as well as increasing renal calcium excretion.

Diuretics may lead to further dehydration. Dialysis may be a last line treatment for life threatening hypercalacemia, but not plasma exchange.

👍 👎 Discuss (4) Improve

Next question

Hypercalcaemia: management ★

The initial management of hypercalcaemia is rehydration with normal saline, typically 3-4 litres/day. Following rehydration bisphosphonates may be used. They typically take 2-3 days to work with maximal effect being seen at 7 days

Other options include:
- calcitonin - quicker effect than bisphosphonates
- steroids in sarcoidosis

Loop diuretics such as furosemide are sometimes used in hypercalcaemia, particularly in patients who cannot tolerate aggressive fluid rehydration. However, they should be used with caution as they may worsen electrolyte derangement and volume depletion.

+ Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 4 👎 7

Hypercalcaemia â€“ presentation and management

Suggest link

Report broken link

Media

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 0 👎 1

Report broken media

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Question 110 of 342

A 23-year-old man with type 1 diabetes presents to the emergency department with vomiting and abdominal pain. He has been suffering from diarrhea for two days and then became severely dehydrated and started to vomit. He has not had his insulin for the last 24 hours. He normally has a basal bolus regime with Levemir as a long-acting insulin, and Humalog as short-acting. He has no other medical problems or regular prescription. On admission he is found to have acidosis, elevated serum ketones and elevated blood glucose, and was diagnosed with diabetic ketoacidosis. He is given rapid fluid infusion. What insulin should he be prescribed?

- ○ Fixed rate IV insulin only ×
- ○ Fixed rate IV insulin as well as long-acting insulin ×
- ○ Fixed rate IV insulin, long-acting insulin and short-acting insulin ×
- ○ Sliding scale IV insulin only ×
- ○ Sliding scale IV insulin as well as long-acting insulin ×

Submit answer

Reference ranges

Score: **56.9%**

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Fixed rate IV insulin only	18%
Fixed rate IV insulin as well as long-acting insulin	72%
Fixed rate IV insulin, long-acting insulin and short-acting insulin	3%
Sliding scale IV insulin only	3%
Sliding scale IV insulin as well as long-acting insulin	4%

In the acute management of DKA, insulin should be fixed rate whilst continuing regular injected long-acting insulin but stopping short actin injected insulin

Important for me Less important

This is a typical presentation for diabetic ketoacidosis in a patient with type 1 diabetes and requires urgent management which should be supported by local guidelines. It is important that IV insulin is started, but any long-acting insulin, Levemir for this patient, should also be continued. This will aid transition back to normal when the DKA resolves and the patient starts to eat and drink again. Giving him short acting insulin is not appropriate during IV insulin therapy which is also a short-acting insulin. Sliding scale IV insulin is no longer recommended in favour of fixed rate insulin.

👍 👎 Discuss (5) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points - glucose > 13.8 mmol/l - pH < 7.30 - serum bicarbonate <18 mmol/l - anion gap > 10 - ketonaemia	Key points - glucose > 11 mmol/l or known diabetes mellitus - pH < 7.3 - bicarbonate < 15 mmol/l - ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids
  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and

- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

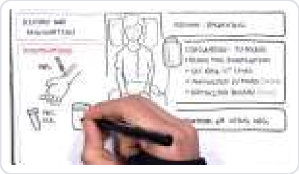
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

Report broken link

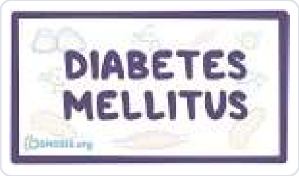
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **57.3%**

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A 45-year-old woman is brought into the resuscitation room with a Glasgow coma scale of 11 (E2 V5 M4). A concerned neighbour called the emergency services, who found them in a moribund state. The neighbour states that she had seemed low over the past couple of months and that she had been wearing more layers of clothes than seemed appropriate.

On initial examination, they feel cool to touch. Pulse is regular and bradycardic with a heart rate of 38 beats per minute. Heart sounds 1+2 are present. Respiratory rate is 8 with oxygen saturations of 91% on 15 L. Auscultation of the chest is clear. Temperature is 33°C. BM is 2.7.

Blood tests return as:

Hb	130 g/L	Male: (135-180) Female: (115 - 160)
Platelets	220 * 10^9/L	(150 - 400)
WBC	9 * 10^9/L	(4.0 - 11.0)

Calcium	2.5 mmol/L	(2.1-2.6)
Thyroid stimulating hormone (TSH)	25 mU/L	(0.5-5.5)
Free thyroxine (T4)	0.4 pmol/L	(9.0 - 18)
Creatine kinase	6000 U/L	(35 - 250)

Na^+	130 mmol/L	(135 - 145)
K^+	4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	8 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)

What is the most appropriate management of this patient?

- ○ Intubation and urgent CT head ✕
- ○ IV hydrocortisone, IV dextrose and rewarming ✕
- ○ IV T4, IV fluids, IV dextrose and intubation ✕
- ○ IV fluid resuscitation, IV dextrose, intubation and IV T4 ✕
- ○ IV hydrocortisone, IV T4, IV dextrose and IV fluids ✕

Submit answer

Reference ranges

Score: **57.3%**

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← Question 111 of 342 ✗ →

A 45-year-old woman is brought into the resuscitation room with a Glasgow coma scale of 11 (E2 V5 M4). A concerned neighbour called the emergency services, who found them in a moribund state. The neighbour states that she had seemed low over the past couple of months and that she had been wearing more layers of clothes than seemed appropriate.

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Creatine kinase	6000 U/L	(35 - 250)

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Bicarbonate	22 mmol/L	(22 - 29)
Urea	8 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)

What is the most appropriate management of this patient?

	Intubation and urgent CT head	1%
🟥	IV hydrocortisone, IV dextrose and rewarming	25%
	IV T4, IV fluids, IV dextrose and intubation	6%
	IV fluid resuscitation, IV dextrose, intubation and IV T4	10%
🟩	IV hydrocortisone, IV T4, IV dextrose and IV fluids	57%

If myxedema coma is suspected, IV corticosteroids should be given alongside IV thyroid replacement until coexisting adrenal insufficiency has been excluded

Important for me Less important

The most appropriate answer is 5 as they need treatment of the underlying cause as well as correction of life-threatening abnormalities. This patient is suffering from severe hypothyroidism and is in a myxedema coma. This is best treated with IV hydrocortisone and thyroxine in case there is co-existing adrenal insufficiency. Their blood sugars are low necessitating correction and there is a raised creatine kinase requiring management with IV fluids.

CT head and intubation is appropriate but is not the best option of those above. The other answers do no include thyroxine and hydrocortisone as a combined answer so, although an aspect of the overall management of the patient, are not the most pressing intervention.

👍 👎 💬 Discuss (6) Improve

Next question

Myxoedema coma ★

Myxoedema coma typically presents with confusion and hypothermia.

Myxoedema coma is a medical emergency requiring treatment with
- IV thyroid replacement
- IV fluid
- IV corticosteroids (until the possibility of coexisting adrenal insufficiency has been excluded)
- electrolyte imbalance correction
- sometimes rewarming

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **56.8%**

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111 ✗

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A 50-year-old obese gentleman presented to the emergency department with sweating, pallor, shortness of breath and central chest pain. He was diagnosed with a STEMI and underwent primary PCI. An ECHO after the event showed an EF of 35%. Following work up for his coronary artery disease he was diagnosed with type 2 diabetes. He was commenced on iv insulin which controlled his blood glucose in the interim.

Investigations

Creatinine	122umol/L
Urea	8.2mmol/l
Na+	140 mmol/l
K+	3.6 mmol/l
eGFR	62 ml/min
HbA1C	9.4%
HCO3	22
Aspartate transaminase	52 U/L
Alkaline phosphatase	110 U/L
Gamma-glutamyl transferase	39 U/L

In the long term treatment of his diabetes which hypoglycaemic agent is best avoided?

- ○ Metformin ×
- ○ Pioglitazone ×
- ○ Gliclazide ×
- ○ Acarbose ×

○ Insulin glargine ×

Submit answer

Reference ranges ⌄

Score: **56.8%**

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112	-

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Back to top

Question 112 of 342 ✗

A 50-year-old obese gentleman presented to the emergency department with sweating, pallor, shortness of breath and central chest pain. He was diagnosed with a STEMI and underwent primary PCI. An ECHO after the event showed an EF of 35%. Following work up for his coronary artery disease he was diagnosed with type 2 diabetes. He was commenced on iv insulin which controlled his blood glucose in the interim.

Investigations

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Na+	140 mmol/l
K+	3.6 mmol/l
eGFR	62 ml/min
HbA1C	9.4%
HCO3	22
Aspartate transaminase	52 U/L
Alkaline phosphatase	110 U/L
Gamma-glutamyl transferase	39 U/L

In the long term treatment of his diabetes which hypoglycaemic agent is best avoided?

	Metformin	7%
🟩	Pioglitazone	82%
🟥	Gliclazide	7%
	Acarbose	3%

Insulin glargine 1%

Pioglitazone - contraindicated by: heart failure

Important for me Less important

Answer: Pioglitazone

One of the side effects of pioglitazone is fluid retention, therefore it is best avoided in a patient with a risk of heart failure like the above patient as it can precipitate or worsen heart failure due to fluid overload.

👍 👎 Discuss (4) Improve

Next question

Thiazolidinediones

Thiazolidinediones are a class of agents used in the treatment of type 2 diabetes mellitus. They are agonists to the PPAR-gamma receptor and reduce peripheral insulin resistance. Rosiglitazone was withdrawn in 2010 following concerns about the cardiovascular side-effect profile.

The PPAR-gamma receptor is an intracellular nuclear receptor. It's natural ligands are free fatty acids and it is thought to control adipocyte differentiation and function.

Adverse effects

- weight gain
- liver impairment: monitor LFTs
- fluid retention - therefore contraindicated in heart failure. The risk of fluid retention is increased if the patient also takes insulin
- recent studies have indicated an increased risk of fractures
- bladder cancer: recent studies have shown an increased risk of bladder cancer in patients taking pioglitazone (hazard ratio 2.64)

＋ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 11 👎 7

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Score: **56.3%**

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112 ✕

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← Question 113 of 342 →

A 24 year-old man presents with a five week history of increasing thirst and frequency of urinating. The GP suspects diabetes and performs two fasting blood tests on separate days which reveal blood glucose results of 8.7 mmol/l and 9.2 mmol/l. Urinalysis does not detect any ketones or protein in the urine. The patient's mother had a diagnosis as type 1 diabetes at the age of 22 and his maternal grandfather and aunt also have type 1 diabetes. Due to the family history, the patient's c-peptide is measured and found to be consistently high on two occasions.

Given the likely diagnosis, what is the most appropriate first treatment for managing this condition?

- ○ Gliclazide ×
- ● Metformin ×
- ○ Pioglitazone ×
- ○ Insulin ×
- ○ Sitagliptin ×

Submit answer

Reference ranges

Score: **56.3%**

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A 24 year-old man presents with a five week history of increasing thirst and frequency of urinating. The GP suspects diabetes and performs two fasting blood tests on separate days which reveal blood glucose results of 8.7 mmol/l and 9.2 mmol/l. Urinalysis does not detect any ketones or protein in the urine. The patient's mother had a diagnosis as type 1 diabetes at the age of 22 and his maternal grandfather and aunt also have type 1 diabetes. Due to the family history, the patient's c-peptide is measured and found to be consistently high on two occasions.

Given the likely diagnosis, what is the most appropriate first treatment for managing this condition?

🟩	Gliclazide	59%
🟥	Metformin	22%
	Pioglitazone	1%
	Insulin	17%
	Sitagliptin	1%

The most likely diagnosis in this scenario is maturity onset diabetes of youth, due to the persistently raised c-peptide and strong family history. It normally presents in early adulthood and 1/3 of cases can be treated with oral hypoglycaemics, such as sulphonylureas. It is therefore important to see the response to sulphonylureas first before commencing insulin therapy.

Discuss (13) | Improve

Next question

MODY ★

Maturity-Onset Diabetes of the Young (MODY) is a form of monogenic diabetes, typically characterized by an autosomal dominant inheritance pattern, onset usually before 25 years of age, and impairment in insulin secretion with minimal or no defects in insulin action. Unlike more common forms of diabetes, such as Type 1 and Type 2, MODY is not primarily driven by lifestyle factors.

It is thought that around 1-2% of patients with diabetes mellitus have MODY, and around 90% are misclassified as having either type 1 or type 2 diabetes mellitus.

Classification and Genetics

MODY is a heterogenous group, with over 14 types identified as of the latest revision, each corresponding to mutations in different genes. The most common types are MODY2 (GCK mutation) and MODY3 (HNF1A mutation), but others include MODY1 (HNF4A), MODY4 (PDX1), MODY5 (HNF1B), and so on.

MODY 3 accounts for around 60% of cases with MODY 2 causing 20% of cases.

Each subtype varies in its clinical presentation, management, and prognosis, emphasizing the importance of precise genetic diagnosis in order to guide treatment strategy and genetic counselling.

Clinical Features

Patients with MODY often present with mild non-ketotic hyperglycemia that is often detected incidentally or during routine screening. It may also be discovered during pregnancy. Unlike Type 1 diabetes, patients with MODY usually do not present with diabetic ketoacidosis except under severe stress conditions, and unlike Type 2 diabetes, they are often of normal weight and do not exhibit signs of insulin resistance.

The specific clinical manifestations and complications can vary depending on the subtype of MODY. For example, individuals with MODY2 generally have mild, stable fasting hyperglycemia and rarely develop severe complications, whereas those with MODY3 or MODY1 may have progressive hyperglycemia and are at higher risk for complications typically associated with diabetes, such as retinopathy, nephropathy, and cardiovascular disease.

Diagnosis

MODY should be suspected in individuals with persistent, asymptomatic hyperglycemia detected before the age of 25, without the typical features of Type 1 or Type 2 diabetes.

The diagnosis is confirmed by genetic testing, which is crucial for identifying the specific type of MODY, as this has direct implications for management. It's important to remember, however, that genetic testing can be expensive and may not be available in all settings.

Treatment

The treatment for MODY depends on the specific genetic subtype. For example, MODY2 often does not require specific treatment, as the hyperglycemia is mild and usually does not lead to complications. In contrast, MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas. Insulin therapy may be necessary in some cases, especially during pregnancy or if sulfonylureas are contraindicated or ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.8%**

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A 17-year-old woman is referred to the endocrinology outpatient clinic with primary amenorrhoea. She has no past medical history and does not take any medications.

On examination, there is no axillary or pubic hair. There are swellings palpable in the groins bilaterally. There is normal breast development.

Testosterone	1100 ng/dl	Male: (300-1200) Female : (20-75)

What is the likely diagnosis?

○ 17-alpha-hydroxylase deficiency ✕

○ Androgen insensitivity syndrome ✕

○ Klinefelter syndrome ✕

○ Pituitary tumour ✕

○ Turner syndrome ✕

Submit answer

Reference ranges

Score: **55.8%**

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Question 114 of 342 ✓

A 17-year-old woman is referred to the endocrinology outpatient clinic with primary amenorrhoea. She has no past medical history and does not take any medications.

On examination, there is no axillary or pubic hair. There are swellings palpable in the groins bilaterally. There is normal breast development.

Testosterone	1100 ng/dl	Male: (300-1200) Female : (20-75)

What is the likely diagnosis?

17-alpha-hydroxylase deficiency	11%
Androgen insensitivity syndrome	78%
Klinefelter syndrome	6%
Pituitary tumour	1%
Turner syndrome	3%

Primary amenorrhoea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome

Important for me Less important

Androgen insensitivity syndrome is the correct answer. This is an X-linked recessive condition caused by end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Patients often present with primary amenorrhoea along with absent pubic/axillary hair. Bilateral groin swellings may reflect undescended testes and breast development may occur as a result of the conversion of testosterone to oestradiol. Testosterone is typically in the upper normal range for a post-pubertal male.

17-alpha-hydroxylase deficiency is incorrect. This is an uncommon form of congenital adrenal hyperplasia that may also present with primary amenorrhoea and external female genitalia. However, in this condition, testosterone will be low as a lack of androgens are characteristic of this disease.

Klinefelter syndrome is incorrect. This is a syndrome where a patient that is phenotypically male has an extra X chromosome (i.e. 47 XXY). Patients may have small testicles and infertility as predominant features. The patient in this scenario is phenotypically female.

Pituitary tumour is incorrect. This may cause delayed puberty if it causes impaired secretion of gonadotrophins (LH and FSH) i.e. hypogonadotropic hypogonadism. However, breast development may also be impaired with a pituitary tumour and testosterone will be in the normal range for a female, in contrast to this case where it is in the high-normal range for a male.

Turner syndrome is incorrect. This can cause primary amenorrhoea as a consequence of primary ovarian insufficiency. Breast development will be abnormal but pubic and axillary hair development will typically be normal. Unlike this case, testosterone will be in the normal range for a female. Patients have a 45XO karyotype.

👍 👎 Discuss (3) Improve

Next question

Androgen insensitivity syndrome ★

Androgen insensitivity syndrome is an X-linked recessive condition due to end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Complete androgen insensitivity syndrome is the new term for testicular feminisation syndrome

Features

- 'primary amenorrhoea'
- little or no axillary and pubic hair
- undescended testes causing groin swellings
- breast development may occur as a result of the conversion of testosterone to oestradiol

Diagnosis

- buccal smear or chromosomal analysis to reveal 46XY genotype
- after puberty, testosterone concentrations are in the high-normal to slightly elevated reference range for postpubertal boys

Management

- counselling - raise the child as female
- bilateral orchidectomy (increased risk of testicular cancer due to undescended testes)
- oestrogen therapy

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **56.1%**

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114	✓

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A 78-year-old man with type 2 diabetes mellitus is reviewed in the diabetes clinic. He is currently taking metformin 1g bd. He also has a history of hypertension and hypothyroidism. His HbA1c one year ago was 44 mmol/mol (6.2%). The most recent test is reported as 46 mmol/mol (6.4%). What is the most appropriate next step in management?

- ○ Increase dose of metformin ×
- ○ Add glimepiride ×
- ○ Add sitagliptin ×
- ○ Add pioglitazone ×
- ○ Make no changes ×

Submit answer

Reference ranges

Score: **56.1%**

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A 78-year-old man with type 2 diabetes mellitus is reviewed in the diabetes clinic. He is currently taking metformin 1g bd. He also has a history of hypertension and hypothyroidism. His HbA1c one year ago was 44 mmol/mol (6.2%). The most recent test is reported as 46 mmol/mol (6.4%). What is the most appropriate next step in management?

Increase dose of metformin	7%
Add glimepiride	4%
Add sitagliptin	9%
Add pioglitazone	1%
[✓ Make no changes]	79%

The correct answer is to **make no changes** to the patient's current management. This is appropriate because the patient's glycaemic control is well within target range for his age and comorbidities. According to UK guidelines (NICE), the target HbA1c for older adults with type 2 diabetes should be individualised, with less stringent targets (up to 64 mmol/mol or 8.0%) being appropriate for those who are frail or elderly. This patient's HbA1c of 46 mmol/mol (6.4%) represents excellent control, and the small increase from 44 mmol/mol (6.2%) over the past year is clinically insignificant. Intensifying treatment would increase the risk of adverse effects without providing meaningful clinical benefit.

Increasing the dose of metformin would be inappropriate as the patient already has good glycaemic control. Increasing the dose would only increase the risk of gastrointestinal side effects and potentially vitamin B12 deficiency without providing significant clinical benefit.

Adding glimepiride, a sulfonylurea, would be inappropriate as it would increase the risk of hypoglycaemia in this elderly patient. Sulfonylureas are particularly risky in older adults due to their potential to cause severe hypoglycaemia, which can lead to falls and cognitive impairment.

Adding sitagliptin, a DPP-4 inhibitor, would be unnecessary given the current level of glycaemic control. While DPP-4 inhibitors have a good safety profile, adding another medication would increase cost and pill burden without clear benefit.

Adding pioglitazone would be inappropriate as thiazolidinediones can cause fluid retention and increase the risk of heart failure. Given this patient's age and existing cardiovascular risk factors (hypertension), pioglitazone would be a poor choice, particularly when current glycaemic control is already adequate.

👍 👎 Discuss (3) Improve

Next question

Diabetes mellitus: management of type 2

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish

- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

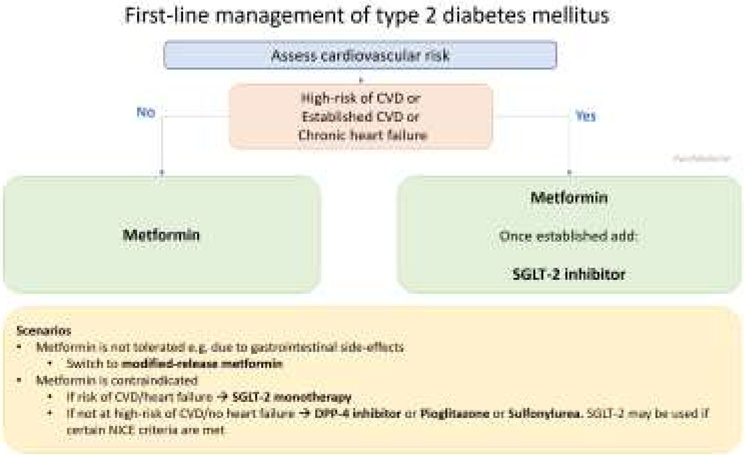
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

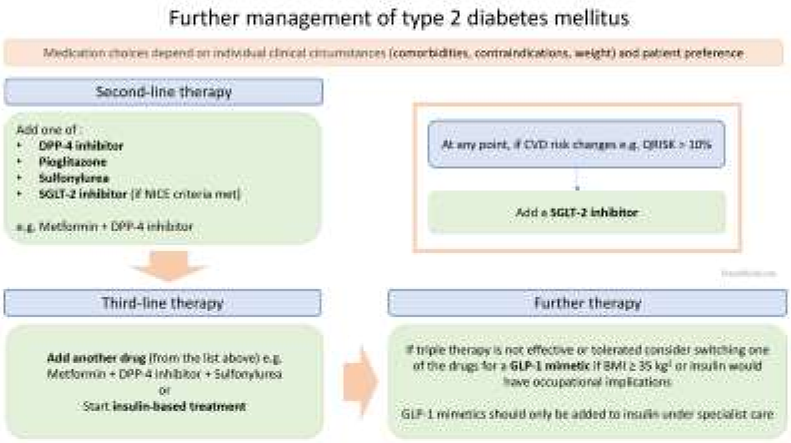
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is

indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

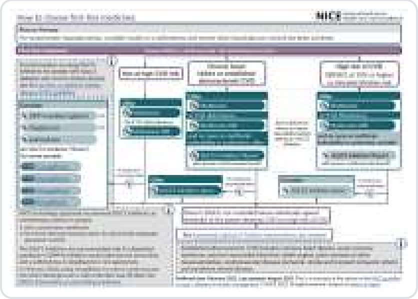
GLP-1 mimetics should only be added to insulin under specialist care

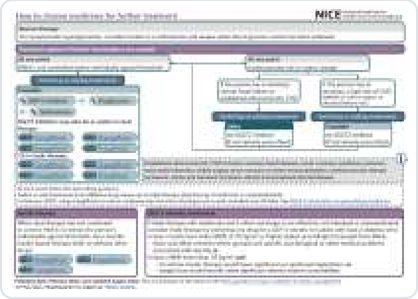
Starting insulin

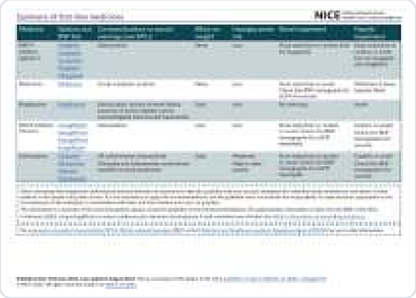
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

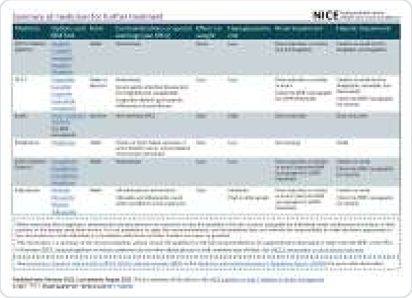
Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea









Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

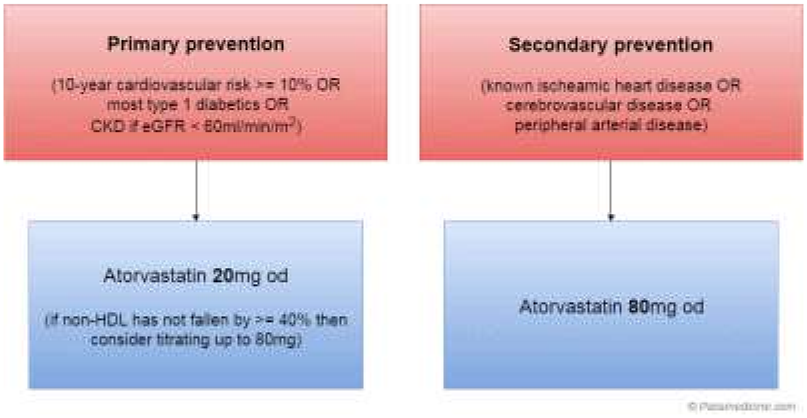
Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be

offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **80**mg od

Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

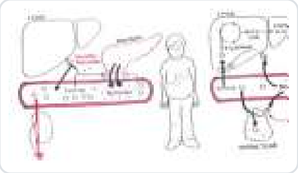
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

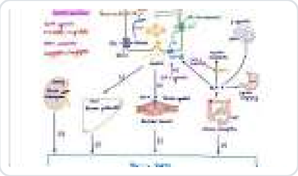
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **56.5%**

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A 19-year-old gentleman with a background history of asthma presents to the Emergency Department complaining of leg weakness and the inability to walk. He had run a marathon the day before. On examination, there is 3/5 weakness of the leg extensors bilaterally. Tone, reflexes and coordination are unimpaired and plantars are downgoing bilaterally. Straight leg raise and sensation to light touch and pain stimulus are unimpaired.

Blood tests show the following:

Hb	13.4g/dl
WBC	6.2 x 10^9/l
Na^+	136mmol/l
K^+	2.9mmol/l
Urea	6.8mmol/l
Creatinine	104µmol/l

What is the most appropriate treatment in this case?

○ Oral potassium and encourage bed rest ✕

○ Hourly forced vital capacity measurements and plasma exchange ✕

○ Oral potassium and encourage gentle exercise ✕

○ Hourly forced vital capacity measurements and IV immunoglobulin ✕

○ Plasma exchange and oral potassium supplementation ✕

Submit answer

Reference ranges

Score: **56.5%**

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Blood tests show the following:

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WBC	6.2 x 10^9/l
Na$^+$	136mmol/l
K$^+$	2.9mmol/l
Urea	6.8mmol/l
Creatinine	104µmol/l

What is the most appropriate treatment in this case?

	Oral potassium and encourage bed rest	45%
	Hourly forced vital capacity measurements and plasma exchange	2%
✓	Oral potassium and encourage gentle exercise	44%
	Hourly forced vital capacity measurements and IV immunoglobulin	5%
	Plasma exchange and oral potassium supplementation	4%

Hypokalaemic periodic paralysis is a rare autosomal dominant periodic paralysis but can also occur as a result of a spontaneous mutation (a third of cases have no family history). Attacks often occur in the morning with a history of strenuous exercise or a high carbohydrate meal the day before or may be provoked by stress eg. infections, lack of sleep. Weakness can range from an isolated muscle group to generalised weakness and tends to affect proximal muscles first.

Serum potassium decreases during attacks but may not necessarily fall below the normal range (3.5 -5mmol/l).

The mainstay of treatment for an acute attack is oral potassium supplementation and encouragement of gentle exercise. Intravenous potassium is reserved for those unable to swallow or with cardiac arrhythmias. Acetazolamide or dichlorphenamide are used as first-line prophylactic agents.

👍 👎 Discuss (17) Improve

Next question

Hypokalaemic periodic paralysis ★

Hypokalaemic periodic paralysis is a rare autosomal dominant disorder characterised by episodes of paralysis, typically occur at night. The underlying defect is a mutation in muscle voltage-gated calcium channels. Attacks may be precipitated by carbohydrate meals

Management
- lifelong potassium supplementation

AI + Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journall 👍 1 👎 1

Review of neurological channelopathies

Suggest link Report broken link

Score: **56.9%**

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Question 117 of 342

A 37-year-old man is being investigated for a chronic metabolic acidosis. He has an anion gap of 9 mEq/L and your consultant suspects he may have a renal source of his acidaemia.

Which of the following can lead to renal tubular acidosis (type 2)?

- ○ Sjogren's syndrome ×
- ○ Wilson's disease ×
- ○ Haemochromatosis ×
- ○ NSAIDs ×
- ○ Nephrocalcinosis ×

Submit answer

Reference ranges

Score: **56.9%**

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117	-

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Back to top

A 37-year-old man is being investigated for a chronic metabolic acidosis. He has an anion gap of 9 mEq/L and your consultant suspects he may have a renal source of his acidaemia.

Which of the following can lead to renal tubular acidosis (type 2)?

	Sjogren's syndrome	18%
🟩	Wilson's disease	59%
	Haemochromatosis	5%
	NSAIDs	12%
🟥	Nephrocalcinosis	6%

Type 2 renal tubular acidosis may be caused by Fanconi syndrome

Important for me Less important

The answer is Wilson's disease.

Renal tubular acidosis (RTA) is a renal cause of a metabolic acidosis. It has 3 main categories:

- Type 1: Distal (failure of proton secretion)
- Type 2: Proximal (failure of bicarbonate reabsorption)
- Type 4: Aldosterone deficiency/insensitivity

Type 3 is a combination of 1 and 2.

Types 1 & 2 are characterised by hypokalaemia and type 4 is characterised by hyperkalaemia. Sjogren's syndrome is one of the classic causes of Type 1 RTA, as well as nephrocalcinosis being both a common cause and consequence of long standing RTA.

NSAIDs lead to aldosterone resistance, causing a Type 4 RTA.

Wilson's disease causes a form of Type 2 RTA called Fanconi syndrome. It can also be caused by other toxic heavy metals (lead & mercury) as well as expired tetracyclines. It leads to proximal loss of protons, phosphate, amino acids and glucose.

Haemochromatosis is a distractor.

👍 👎 Discuss (2) Improve

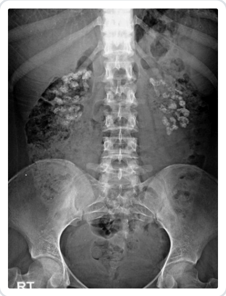
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia

- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

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A 35-year-old female presented to the emergency department with severe right flank pain radiating to her groin. She had recently been referred to the rheumatology department by her GP for investigation of joint pains, dry eyes and dry mouth. She was not taking any regular medication.

On examination her blood pressure was 132/68 mmHg. Abdominal examination revealed right flank tenderness. Blood tests are shown below:

Na^+	136 mmol/L	(135 - 145)
K^+	2.8 mmol/L	(3.5 - 5.0)
Urea	3.6 mmol/L	(2.0 - 7.0)
Creatinine	70 µmol/L	(55 - 120)
Bicarbonate	9 mmol/L	(22 - 28)
Chloride	116 mmol/L	(95 - 105)
Calcium	2.3 mmol/L	(2.1-2.6)
Phosphate	1.1 mmol/L	(0.8-1.4)

What is the most likely diagnosis?

○ Bartter syndrome ×

○ Fanconi syndrome ×

○ Gitelman syndrome ×

○ Lactic acidosis ×

○ Distal renal tubular acidosis ×

Submit answer

Reference ranges ⌄

Score: **56.4%**

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118	-

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A 35-year-old female presented to the emergency department with severe right flank pain radiating to her groin. She had recently been referred to the rheumatology department by her GP for investigation of joint pains, dry eyes and dry mouth. She was not taking any regular medication.

On examination her blood pressure was 132/68 mmHg. Abdominal examination revealed right flank tenderness. Blood tests are shown below:

Na^+	136 mmol/L	(135 - 145)
K^+	2.8 mmol/L	(3.5 - 5.0)
Urea	3.6 mmol/L	(2.0 - 7.0)
Creatinine	70 µmol/L	(55 - 120)
Bicarbonate	9 mmol/L	(22 - 28)
Chloride	116 mmol/L	(95 - 105)
Calcium	2.3 mmol/L	(2.1-2.6)
Phosphate	1.1 mmol/L	(0.8-1.4)

What is the most likely diagnosis?

	Bartter syndrome	4%
🟥	Fanconi syndrome	11%
	Gitelman syndrome	7%
	Lactic acidosis	1%
🟩	Distal renal tubular acidosis	78%

Distal renal tubular acidosis can cause calcium phosphate renal stones and is linked to Sjogrens syndrome

Important for me Less important

This patient presents with renal colic. Blood tests show severe metabolic acidosis and hypokalaemia which fits with a distal renal tubular acidosis (RTA). The history of dry eyes and mouth is suggestive of Sjogrens syndrome, which is frequently associated with distal RTA. Patients with distal RTA are at risk of developing calcium phosphate stones.

Fanconi syndrome causes a proximal RTA which tends to cause milder acidosis and hypokalaemia, along with other electrolyte disturbances such as hypophosphataemia.

Gitelman and Bartter syndromes cause a hypokalaemic alkalosis and are therefore incorrect.

Lactic acidosis would cause a raised anion gap acidosis, whereas the anion gap in this case is normal (12).

👍 👎 Discuss (3) Improve

Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy

Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

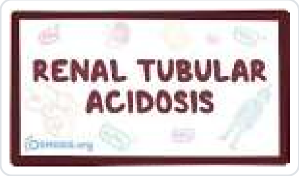
Next question

Textbooks

High-yield textbook

Extended textbook

Media


RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

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Score: **55.9%**

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A 25 year old woman presents to the endocrinology clinic. She is concerned because her father had a 'brain tumour' removed 2 years ago and has now been told he has another tumour in his abdomen after going to his doctor with reflux and indigestion. He has been told it might be a genetic problem and is awaiting testing. She is concerned she might also have the condition and so her GP has referred her to the clinic.

She is currently asymptomatic.

On examination there is no abnormality on the cardiovascular, respiratory, abdominal or neurological examinations.

The doctor explain that the most appropriate person to see would be the geneticist to whom her father has been referred. He asks her to obtain the details if her father is willing to provide them and says that he will refer her. In the meaning time he offers to carry out some screening blood tests.

Given the likely underlying diagnosis, which of the following is most likely to be abnormal?

- ○ Cortisol ×
- ○ Fasting glucose ×
- ○ Parathyroid hormone ×
- ○ Prolactin ×
- ○ Thyroid stimulating hormone ×

Submit answer

Reference ranges ⌄

Score: **55.9%**

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Question 119 of 342 ✗

A 25 year old woman presents to the endocrinology clinic. She is concerned because her father had a 'brain tumour' removed 2 years ago and has now been told he has another tumour in his abdomen after going to his doctor with reflux and indigestion. He has been told it might be a genetic problem and is awaiting testing. She is concerned she might also have the condition and so her GP has referred her to the clinic.

She is currently asymptomatic.

On examination there is no abnormality on the cardiovascular, respiratory, abdominal or neurological examinations.

The doctor explain that the most appropriate person to see would be the geneticist to whom her father has been referred. He asks her to obtain the details if her father is willing to provide them and says that he will refer her. In the meaning time he offers to carry out some screening blood tests.

Given the likely underlying diagnosis, which of the following is most likely to be abnormal?

	Cortisol	3%
🟥	Fasting glucose	7%
🟩	Parathyroid hormone	71%
	Prolactin	15%
	Thyroid stimulating hormone	3%

This lady's father is likely to have multiple endocrine neoplasia type 1 (MEN1) as evidenced by 2 MEN1 associated tumours, a pituitary adenoma and a gastrinoma.

Although this lady is asymptomatic, it is important she is offered genetic screening, as the condition is autosomal dominant.

Fasting glucose, parathyroid hormone and prolactin are all biochemical screening tests used for MEN1 associated tumours (insulinoma, parathyroid adenoma and pituitary adenoma respectively). However, hyperparathyroidism is by far the most common initial manifestation and will eventually develop in 90% of MEN1 patients.

ReferencThakker et al. Clinical guidelines for multiple endocrine neoplasia type 1 (MEN1). J Clin Endocrinol Metab. 2012:97(9)2990-3011)

👍 👎 Discuss (8) Improve

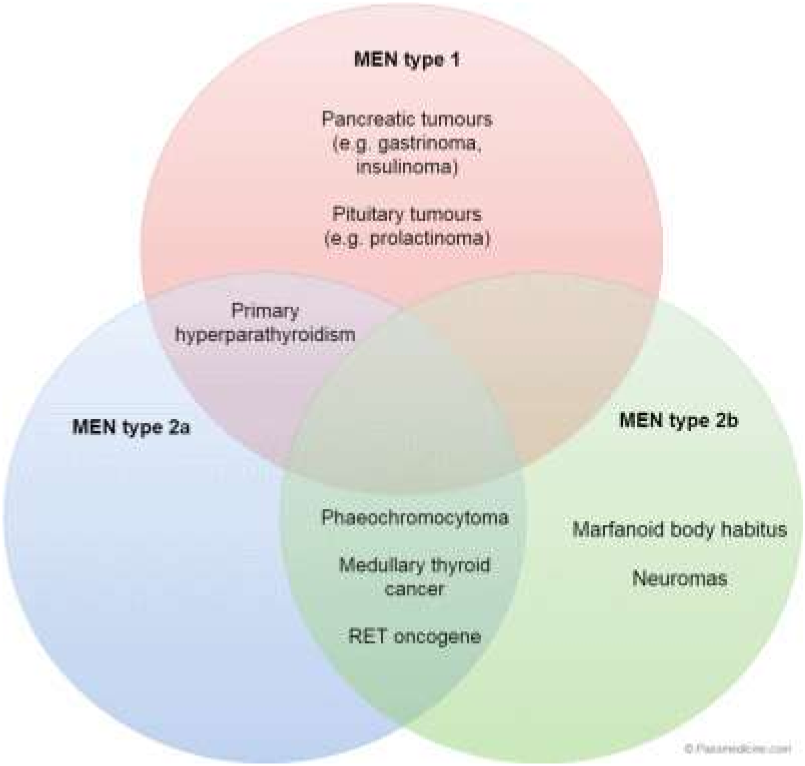
Next question

Multiple endocrine neoplasia ★

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas

MEN type I	MEN type IIa	MEN type IIb
ulceration) Also: adrenal and thyroid		
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
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Venn diagram showing the different types of MEN and their associated features

Q 123

Next question

Textbooks

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Extended textbook

Media

Pancreatic
Neuroendocrine
Neoplasms

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **55.5%**

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Question 120 of 342

A 55 year-old female presents to the outpatients department having been referred by her GP. She complains of fatigue, increased sweating and weight loss over the past four months. She also reports a loss of sex drive.

Examination reveals that she is pale and has a pulse rate of 121 per minute with a bounding pulse character. Her blood pressure is 118/79 mmHg and she has heart sounds 1 and 2 presents with no added sounds. On auscultation, her chest is clear and her abdomen is soft and non-tender with no organomegaly. She has a smooth goitre but has no signs of thyroid eye disease. Examination of her cranial nerves are normal.

The results of recent blood tests are as follows:

Hb	11.3 g/dl
Platelets	190 * 10^9/l
WBC	10.9 * 10^9/l
Na^+	129 mmol/l
K^+	4.3 mmol/l
Urea	7.9 mmol/l
Creatinine	94 µmol/l
ALP	155 u/l
Calcium	2.40 mmol/l
Albumin	40 g/L
TSH	11 mU/L
Free T4	41 pmol/L
Free T3	11 pmol/L

Which of the following is the most likely diagnosis?

○ Grave's disease ×

○ Thyroid cancer ×

○ Surreptitious thyroxine ingestion ×

○ De Quervain's thyroiditis ×

○ TSH secreting pituitary tumour ×

Submit answer

Reference ranges ∨

Score: **55.5%**

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A 55 year-old female presents to the outpatients department having been referred by her GP. She complains of fatigue, increased sweating and weight loss over the past four months. She also reports a loss of sex drive.

Examination reveals that she is pale and has a pulse rate of 121 per minute with a bounding pulse character. Her blood pressure is 118/79 mmHg and she has heart sounds 1 and 2 presents with no added sounds. On auscultation, her chest is clear and her abdomen is soft and non-tender with no organomegaly. She has a smooth goitre but has no signs of thyroid eye disease. Examination of her cranial nerves are normal.

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Hb	11.3 g/dl
Platelets	190 * 10^9/l
WBC	10.9 * 10^9/l
Na$^+$	129 mmol/l
K$^+$	4.3 mmol/l
Urea	7.9 mmol/l
Creatinine	94 µmol/l
ALP	155 u/l
Calcium	2.40 mmol/l
Albumin	40 g/L
TSH	11 mU/L
Free T4	41 pmol/L
Free T3	11 pmol/L

Which of the following is the most likely diagnosis?

	Grave's disease	14%
	Thyroid cancer	5%
🟥	Surreptitious thyroxine ingestion	14%
	De Quervain's thyroiditis	3%
🟩	TSH secreting pituitary tumour	64%

Biochemistry reveals elevated thyroid-stimulating hormone (TSH) with concurrent elevated thyroxine (T4) and tri-iodothyronine (T3). An elevated alkaline phosphatase (ALP) is consistent with thyrotoxicosis. Hyponatraemia suggests hypoadrenalism.

Taken with the symptoms, this patient has a likely diagnosis of a thyrotropinoma, which is a rare type of pituitary tumour accounting for approximately less than 1% of cases of pituitary tumours. 90% are macroadenomas.

Presentation is typically with features of thyrotoxicosis and include weight loss, sweating, fatigue and tachycardia. There may also be signs of hypopituitarism.

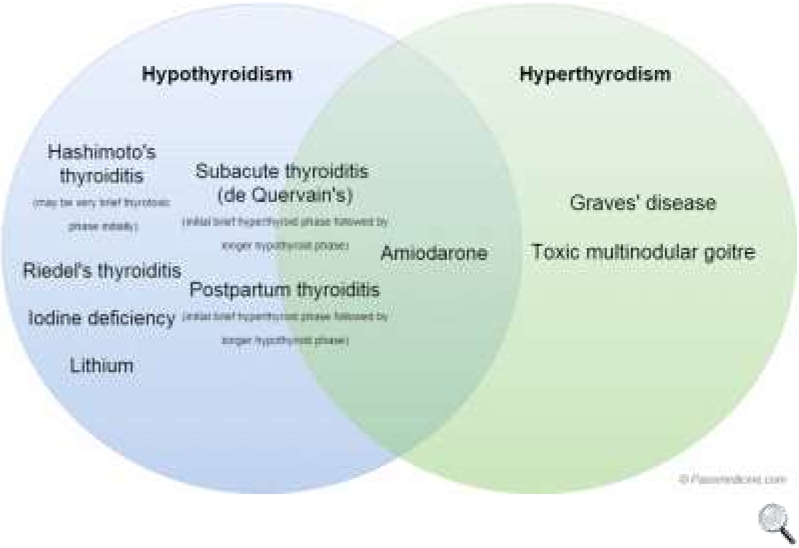
Discuss (6) | Improve

Next question

Thyroid function tests ★

The interpretation of thyroid function tests is usually straightforward:

Diagnosis	TSH	Free T4	Notes
Thyrotoxicosis (e.g. Graves' disease)	Low	High	In T3 thyrotoxicosis the free T4 will be normal
Primary hypothyroidism (primary atrophic hypothyroidism)	High	Low	
Secondary hypothyroidism	Low	Low	Replacement steroid therapy is required prior to thyroxine
Sick euthyroid syndrome*	Low**	Low	Common in hospital inpatients T3 is particularly low in these patients
Subclinical hypothyroidism	High	Normal	
Poor compliance with thyroxine	High	Normal	
Steroid therapy	Low	Normal	


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

*now referred to as non-thyroidal illness
**TSH may be normal in some cases

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

7 Minute Medics Podcast 👍 11 👎 7

Thyroid Disorders

Suggest link Report broken link

Media

THYROID
FUNCTION
TESTS

Understanding Thyroid Function Tests

Zero To Finals - YouTube 👍 0 👎 0

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Score: **55%**

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← Question 121 of 342 →

A 22-year-old woman with a history of partial Kallmann syndrome comes to the fertility clinic for review. She got married some 6 months earlier and wants to start a family. She has normal external genitalia and sparse pubic and axillary hair and has a normal body mass index of 23kg/m². Which of the following is the most appropriate intervention?

- ○ Clomiphene ✕
- ○ HCG and FSH then IVF ✕
- ○ Metformin ✕
- ○ Oestrogen ✕
- ○ Referral for adoption ✕

Submit answer

Reference ranges

Score: **55%**

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A 22-year-old woman with a history of partial Kallmann syndrome comes to the fertility clinic for review. She got married some 6 months earlier and wants to start a family. She has normal external genitalia and sparse pubic and axillary hair and has a normal body mass index of 23kg/m². Which of the following is the most appropriate intervention?

	Clomiphene	18%
🟩	HCG and FSH then IVF	57%
	Metformin	1%
🟥	Oestrogen	8%
	Referral for adoption	16%

The correct answer is HCG and FSH then IVF. Restoration of ovulation in females with Kallmann syndrome is complex and often requires HCG to drive production of gonadal steroid hormones, FSH to drive ovulation, harvesting of eggs, and IVF. This process is most effective in achieving successful pregnancy.

Clomiphene does induce ovulation and is useful in patients with other conditions such as polycystic ovarian syndrome. It is however very unlikely to be effective in patients with gonadotrophin failure like Kallmann syndrome. Metformin has previously been used in the management of PCOS. Oestrogen doesn't restore ovulation in patients with Kallmann syndrome but may be considered for patients who don't want to get pregnant. In the event that fertility treatment isn't successful, adoption can be considered.

Discuss (4) | Improve

Next question

Kallmann's syndrome

Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. It is usually inherited as an X-linked recessive trait. Kallmann's syndrome is thought to be caused by failure of GnRH-secreting neurons to migrate to the hypothalamus.

The clue given in many questions is lack of smell (anosmia) in a boy with delayed puberty.

Features

- 'delayed puberty'
- hypogonadism, cryptorchidism
- anosmia
- sex hormone levels are low
- LH, FSH levels are inappropriately low/normal
- patients are typically of normal or above-average height

Cleft lip/palate and visual/hearing defects are also seen in some patients

Management

- testosterone supplementation
- gonadotrophin supplementation may result in sperm production if fertility is desired later in life

+ Q 123

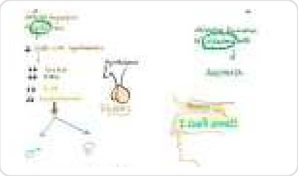
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Kallmann Syndrome mnemonic

Medicosis Perfectionalis - YouTube 👍 0 👎 0

KALLMANN
SYNDROME

Kallman's syndrome

Osmosis - YouTube 👍 8 👎 2

Report broken media

Score: **54.5%**

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A 16-year-old male presents with 3 months of chronic headaches and visual blurring. He has no past medical history and no known family history. On examination, his heart sounds are normal with no added sounds and the respiratory examination is unremarkable. He has no focal neurological signs. Fundoscopy reveals papilloedema, hard exudates and flame haemorrhage. His blood pressure is 226/160mmHg. His blood tests and arterial blood gas are as follows:

Na^+	145 mmol/l
K^+	2.9 mmol/l
Urea	5.4 mmol/l
Creatinine	72 µmol/l

pH	7.49
PaO2	13kPa
PaCO2	3.4 kPa
Bicarbonate	34 mmol/L

Serum ambulatory renin activity	0.2 pmol/L @ 3-4 hours (normal range 0.8-3.5 pmol/ml/hr)
Serum ambulatory aldosterone	24 pmol/L@ 3-4 hours (normal range 100-800)

What is the optimal long-term treatment?

○ Amlodipine ×

○ Ramipril ×

○ Atenolol ×

○ Doxazosin ×

○ Amiloride ×

Submit answer

Reference ranges ∨

Score: **54.5%**

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Question 122 of 342 ✗

A 16-year-old male presents with 3 months of chronic headaches and visual blurring. He has no past medical history and no known family history. On examination, his heart sounds are normal with no added sounds and the respiratory examination is unremarkable. He has no focal neurological signs. Fundoscopy reveals papilloedema, hard exudates and flame haemorrhage. His blood pressure is 226/160mmHg. His blood tests and arterial blood gas are as follows:

Na^+	145 mmol/l
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Serum ambulatory renin activity	0.2 pmol/L @ 3-4 hours (normal range 0.8-3.5 pmol/ml/hr)
Serum ambulatory aldosterone	24 pmol/L@ 3-4 hours (normal range 100-800)

What is the optimal long-term treatment?

Amlodipine	3%
Ramipril	10%
Atenolol	2%
Doxazosin	16%

Amiloride 69%

The patient is young and presents with grade 4 hypertensive retinopathic changes associated with a systolic of over 200mmHg, associated with hypokalaemia and a metabolic alkalosis. Importantly, both renin and aldosterone are decreased, ruling out primary hyperaldosteronism. The diagnosis is thus likely to be Liddle's syndrome, a genetic disorder of ENaC channels in the collecting duct, leading to increased sodium reabsorption and increased potassium excretion. The treatment is with amiloride, a potassium sparing diuretic that directly blocks collecting tubule sodium channels and resolves hypertension.

👍 👎 Discuss (7) Improve

Next question

Liddle's syndrome ★

Liddle's syndrome is a rare autosomal dominant condition that causes hypertension and hypokalaemic alkalosis. It is thought to be caused by disordered sodium channels in the distal tubules leading to increased reabsorption of sodium.

Treatment is with either amiloride or triamterene

+ Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Media

LIDDLE'S SYNDROME
The Medics Guide

Liddle's syndrome

The Medics Guide - YouTube 👍 16 👎 0

Report broken media

Score: **54.1%**

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You are asked to review a 67-year-old man who is currently an inpatient on a surgical ward with new paraesthesia in his fingers. He was admitted for an elective parathyroidectomy three days ago for fairly long standing hyperparathyroidism and subsequent hypercalcaemia. He had a single parathyroid adenoma excised which had been identified on pre-operative MIBI scanning. The procedure was without complications but he is now complaining of a tingling sensation in his fingers that he first noticed about twelve hours ago. He also complains of new severe pain in both of his ankles which is worse when he walks, but also present at rest. The surgical SHO has already arranged x-rays of the patient's ankles and these reveal multiple osteolytic lesions which have been reported as being suspicious for metastatic disease. He is otherwise fit and well and his only regular medications are paracetamol, tramadol and prophylactic dalteparin. His blood tests are as follows.

Adjusted Calcium	1.84 mmol/L
Magnesium	0.7 mmol/L

What is the most likely explanation for his current symptoms?

- ○ Metastatic parathyroid cancer ×
- ○ Secondary hyperparathyroidism ×
- ○ Hypomagnesaemia ×
- ○ Hungry bone syndrome ×
- ○ Secondary hypoparathyroidism ×

Submit answer

Reference ranges ⌄

Score: **54.1%**

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Back to top

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Adjusted Calcium	1.84 mmol/L
Magnesium	0.7 mmol/L

What is the most likely explanation for his current symptoms?

	Metastatic parathyroid cancer	3%
	Secondary hyperparathyroidism	3%
🟥	Hypomagnesaemia	11%
🟩	Hungry bone syndrome	74%
	Secondary hypoparathyroidism	9%

Hypocalcaemia after parathyroid surgery is relatively common and usually 'benign' and associated with a transient hypoparathyroidism. However, it can sometimes be more marked and give rise to symptoms such as perioral or finger paraesthesia. This state alone would not, however, explain his ankle pain or x-ray findings.

Although hypomagnesaemia may also be present and should be treated, it does not explain the symptoms. Metastatic parathyroid cancer is a possibility given the x-ray findings, but is very uncommon and is less likely given that his hyperparathyroidism and hypercalcaemia was long standing (i.e. indolent). Secondary hyperparathyroidism is the syndrome of appropriately raised parathyroid hormone in response to hypocalcaemia, usually secondary to chronic kidney disease. Secondary hypoparathyroidism describes the normal parathyroid hormone suppression that occurs in hypercalcaemia secondary to non-parathyroid causes, such as malignancy.

Discuss (3) | Improve

Next question

Hungry bone syndrome

Hungry bone syndrome is an uncommon entity but can occur after parathyroidectomy if the hyperparathyroidism has been long-standing. The mechanism is thought to be thus: high pre-operative levels of parathyroid hormone provide a constant stimulus for osteoclast activity creating the hypercalcaemic state by de-mineralizing the bones. This process can result in x-ray changes very similar to metastatic lytic lesions if left untreated. Upon removal of the parathyroid adenoma the hormone levels fall rapidly (they have a very short half-life) and the osteoclast activity is subsequently diminished and the bones rapidly begin re-mineralisation - 'hungry bone syndrome'. This process can be uncomfortable and also result in systemic hypocalcaemia.

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **53.7%**

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A 40 year old man presents to the Emergency Department with tiredness and dizziness (worse on standing) which has been ongoing for the past few months. He had a past medical history of epilepsy and mentions that he has had 'brain surgery' in the past. He is on some medications but cannot remember the names. He has no allergies.

On assessment, he has no focal neurological deficit and cardiovascular/respiratory examination is normal. Observations show a blood pressure of 135/90 mmHg (dropping to 105/82 mmHg on standing), a heart rate of 67 beats per minute, a temperature of 36.2 degrees, oxygen saturations of 94% on air and a respiratory rate of 18/min. Given his medical history, you opt to keep this gentleman in the short stay unit for observation overnight.

Baseline blood tests are as follows:

Hb	125 g/l
WCC	9.2 x109/l
Plt	290 x109/l
CRP	10 mg/l
Gluc	3.9 mmol/l
Na+	138 mmol/l
K+	5.8 mmol/l
Ur	7.2 mmol/l
Cr	100 µmol/l
TSH	0.4 mU/l
T4	5.0 pmol/l

Given the above, what is the most likely underlying diagnosis?

○ Hypopituitarism ✕

○ Hypothyroidism ×

○ Acromegaly ×

○ Pheochromocytoma ×

○ Medication side effects ×

Submit answer

Reference ranges

Score: **0%**

1 -

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Question 1 of 219 ✓

A 40 year old man presents to the Emergency Department with tiredness and dizziness (worse on standing) which has been ongoing for the past few months. He had a past medical history of epilepsy and mentions that he has had 'brain surgery' in the past. He is on some medications but cannot remember the names. He has no allergies.

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K+	5.8 mmol/l
Ur	7.2 mmol/l
Cr	100 µmol/l
TSH	0.4 mU/l
T4	5.0 pmol/l

Given the above, what is the most likely underlying diagnosis?

Hypopituitarism 82%

Hypothyroidism	4%
Acromegaly	1%
Pheochromocytoma	2%
Medication side effects	11%

This gentleman has hypopituitarism following 'brain surgery'. Though the details of this are obscured in the question, it is likely that removal of a pituitary mass with trans-sphenoidal surgery. This is exhibited by fairly non-descript symptoms coupled with some underlying evidence of lack of anterior pituitary hormones: low BP/dizziness/postural hypotension, high/normal K+, low/normal Na+ and low/normal blood glucose all indicate lack of cortisol due to low ACTH; the low/normal temperature and heart rate and the tiredness steer you towards low thyroxine level due to lack of TSH.

This gentleman needs assessment of his pituitary function. This can be done in many ways. A baseline pituitary hormone profile can be quite useful; however the most definitive tests involve assessing dynamic pituitary function. The insulin stress test (coupled with TRH and GnRH tests) creates a hypoglycaemic effect in the body and the response of the pituitary (cortisol surge) is measured. However, inducing hypoglycaemia in epileptics, such as this gentleman, is contraindicated. Therefore the next best investigation is the glucagon stimulation test which mimics hypoglycaemia in the body and causes a fake stress on the pituitary, therefore being safe to use in epileptics.

Discuss (4) Improve

Next question

Hypopituitarism

Hypopituitarism describes the inadequate production of one or more of the hormones secreted by the pituitary gland.

Causes

- compression of the pituitary gland by non-secretory pituitary macroadenoma (most common)
- pituitary apoplexy
- Sheehan's syndrome: postpartum pituitary necrosis secondary to a postpartum haemorrhage
- hypothalamic tumours e.g. craniopharyngioma
- trauma
- iatrogenic irradiation
- infiltrative e.g. hemochromatosis, sarcoidosis

The clinical features depend on which hormones are deficient:

- low ACTH
  - tiredness
  - postural hypotension
- low FSH/LH
  - amenorrhoea
  - infertility
  - loss of libido
- low TSH
  - feeling cold
  - constipation
- low GH
  - if occurs during childhood then short stature
- low prolactin
  - problems with lactation
- there may also be features suggestive of the underlying causes
  - pituriary macroadenoma → bitemporal hemianopia
  - pituitary apoplexy → sudden, severe headache

Investigations

- hormone profile testing
- imaging

Management

- treatment of any underlying cause (e.g. surgical removal of pituitary macroadenoma)

- replacement of deficient hormones

+ Q 123

Next question

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Media

HYPOPITUITARISM

Hypopituitarism

Osmosis - YouTube 👍 5 👎 0

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1 ✓

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A 23-year-old female presented with acne and hirsutism worsening over the last 3 years. She attained menarche aged 10 and has irregular periods.

On examination, body mass index 29kg/m², heart rate 80/min, blood pressure 135/85 mmHg. Hirsutism and acanthosis nigricans are noticed along with mild clitoromegaly.

Bloods on 6th day after menstruation:

Estradiol	300 pmol/L (early follicular NR<300 pmol/L)
17OH-progesterone	20 nmol/L (NR<10 nmol/L)
Free Testosterone	3 nmol/L (NR<3 nmol/L)
LH	4 IU/L (NR 1-9 IU/L)
FSH	3 IU/L (NR 1-13 IU/L)
9am cortisol	150 nmol/L (NR 200-700 nmol/L)

What is the single most useful test?

- ○ CT adrenals ×
- ○ Karyotype ×
- ○ Pelvic USS ×
- ○ Short synacthen test ×
- ○ MRI pituitary ×

Submit answer

Reference ranges

Score: **100%**

1	✓
2	-

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LH	4 IU/L (NR 1-9 IU/L)
FSH	3 IU/L (NR 1-13 IU/L)
9am cortisol	150 nmol/L (NR 200-700 nmol/L)

What is the single most useful test?

CT adrenals	24%
Karyotype	21%
Pelvic USS	29%
[✓ Short synacthen test]	20%
MRI pituitary	6%

ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

Important for me Less important

This patient has hyperandrogenism (acne, hirsutism, high 17OH-progesterone) indicative of non-classical congenital adrenal hyperplasia.

CAH is caused by:

1. 21 hydroxylase mutation (90% of CAH).

This mediates conversion of 17OH-progesterone to 11-deoxycortisol and progesterone to deoxycorticosterone. Loss of function results in reduced cortisol and aldosterone and subsequent elevation in ACTH. This causes overstimulation of the adrenal cortex (hyperplasia). The steroid precursors are forced down the sex hormone pathway leading to androgen excess (ambiguous genitalia, salt wasting, hypovolaemia and shock i.e classic type). Presentation of 21 hydroxylase deficiency can also be asymptomatic with androgen excess become a problem in late childhood i.e. non-classic (premature pubarche, accelerated bone age, acne, hirsutism, oligomenorrhoea and this mimics polycystic ovarian syndrome).

2. 11-beta hydroxylase mutation (5% of CAH).

Raised BP as 11-deoxycortisol has some aldosterone activity. Also raised androgens.

3. 17-alpha hydroxylase deficiency

Raised aldosterone but low androgens.

4. 3-beta steroid dehydrogenase

Low aldosterone and low androgens.

The short synacthen test is helpful when 17OH-progesterone is only modestly elevated (if 17OH-progesterone is very high then it is diagnostic). Measuring 17OH-progesterone at 0 and 60 mins after ACTH causes elevated responses in patients with CAH (>35 nmol/L).

Discuss (9) | Improve

Next question

Congenital adrenal hyperplasia

Congenital adrenal hyperplasia (CAH) refers to a group of autosomal recessive disorders that impair adrenal steroid biosynthesis. The deficiency in cortisol production leads to compensatory overproduction of adrenocorticotropic hormone (ACTH) by the anterior pituitary. Elevated ACTH levels increase the production of adrenal androgens, which can result in the virilization of female infants and affect genital development.

Cause

- 21-hydroxylase deficiency (90%)
  - impairs the conversion of 17-hydroxyprogesterone to 11-deoxycortisol, leading to cortisol deficiency and excess androgen production
- 11-beta hydroxylase deficihttps://www.passmedicine.com/database/dbnotes.php#ency (5%)
  - results in hypertension due to excess deoxycorticosterone
- 17-hydroxylase deficiency (very rare)
  - leads to mineralocorticoid excess with low androgen and estrogen levels

Clinical features

- the symptoms and signs of CAH vary depending on the specific enzyme deficiency and the severity of the disorder. Common clinical presentations include:
- virilization:
  - female infants may present with ambiguous genitalia due to excessive androgen exposure in utero.
  - male infants appear normal at birth, which can delay diagnosis.
- salt-wasting crisis:

  - this severe form occurs in about 75% of cases with 21-hydroxylase deficiency
  - it is characterized by dehydration, hypotension, and electrolyte imbalances, and can be life-threatening if not treated promptly.
- precocious puberty: Excess androgens can lead to early development of secondary sexual characteristics in both males and females.
- infertility: Adults with untreated CAH may experience fertility issues due to hormonal imbalances.
- height and growth abnormalities: Children with CAH often experience accelerated growth rates initially but may have a shorter adult stature due to early epiphyseal closure.

Diagnosis
- many countries screen newborns for CAH, looking for elevated levels of serum concentration of 17-hydroxyprogesterone (17OHP). This is however not yet done in the UK
- ACTH stimulation testing is used to confirm the diagnosis
  - evaluates the adrenal gland's response to ACTH, with abnormal increases in 17OHP indicating CAH

Management
- primarily involves glucocorticoid replacement to reduce ACTH levels and minimize adrenal androgen production
- in cases of mineralocorticoid deficiency fludrocortisone is also prescribed

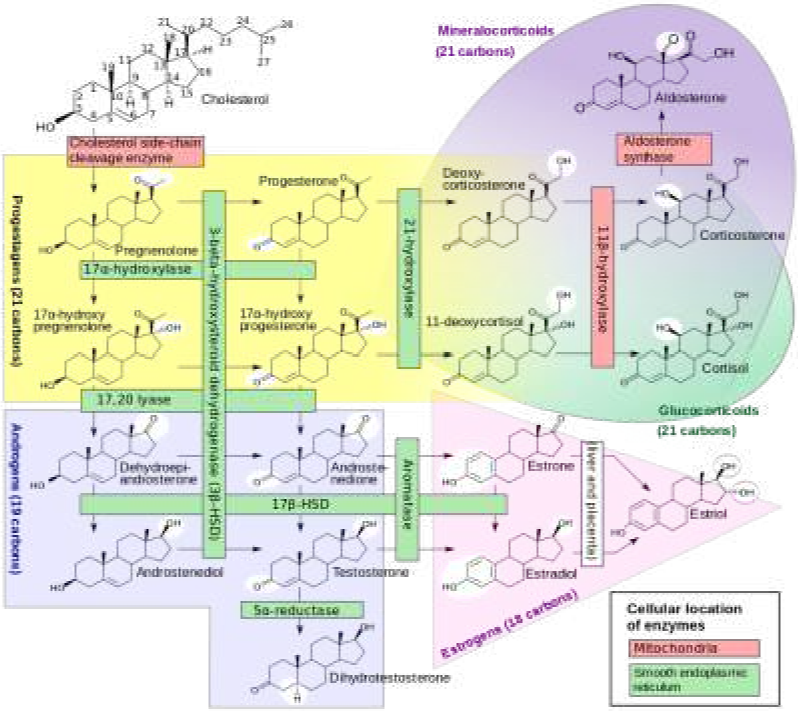
Cholesterol
HO
Cholesterol side-chain cleavage enzyme
Mineralocorticoids (21 carbons)
Aldosterone
Aldosterone synthase
Progestagens (21 carbons)
Pregnenolone
Progesterone
Deoxy-corticosterone
Corticosterone
17α-hydroxylase
3-beta-hydroxysteroid dehydrogenase (3β-HSD)
21-hydroxylase
11β-hydroxylase
17α-hydroxy pregnenolone
17α-hydroxy progesterone
11-deoxycortisol
Cortisol
Glucocorticoids (21 carbons)
17,20 lyase
Androgens (19 carbons)
Dehydroepi-androsterone
Androste-nedione
Aromatase
Estrone
liver and placenta
Estriol
17β-HSD
Androstenediol
Testosterone
Estradiol
5α-reductase
Dihydrotestosterone
Estrogens (18 carbons)
Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

Q 123

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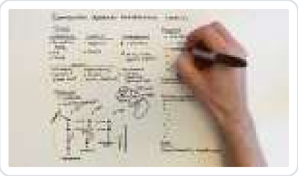
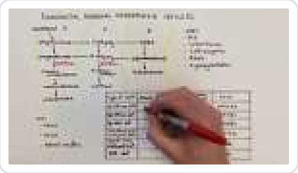


Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 👍 18 👎 2



Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 👍 13 👎 2

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2 ✓

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A lady who is 10 weeks pregnant presents to her antenatal appointment asking for advice regarding gestational diabetes. She is a 31-year-old white woman with a BMI (body mass index) of 28.7. In terms of family history, she has a cousin who has type 1 diabetes mellitus and an aunt who is being treated for breast cancer. She has had two previous pregnancies, the first one she, unfortunately, miscarried at 8 weeks, and the second was a normal pregnancy that she took to term with a birth weight of 4.6kg. Neither of these pregnancies was complicated by gestational diabetes, and the baby is now 2 years old and has not had to be taken to see a doctor other than routine appointments.

What is the most appropriate testing regime for ruling out gestational diabetes in this woman?

- ○ Oral glucose tolerance test at 24-28 weeks pregnant ✕
- ○ None - as she has no risk factors for gestational diabetes ✕
- ○ Oral glucose tolerance test at 12-14 weeks pregnant ✕
- ○ Self-monitoring of sugars and repeat appointment in 2 weeks ✕
- ○ HBa1c ✕

Submit answer

Reference ranges

Score: **0%**

1	-

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What is the most appropriate testing regime for ruling out gestational diabetes in this woman?

🟩	Oral glucose tolerance test at 24-28 weeks pregnant	52%
	None - as she has no risk factors for gestational diabetes	2%
🟥	Oral glucose tolerance test at 12-14 weeks pregnant	39%
	Self-monitoring of sugars and repeat appointment in 2 weeks	5%
	HBa1c	1%

This question requires knowledge on the risk factors for developing gestational diabetes and appropriate testing based on risk:

Risk factors for gestational diabetes include:

- BMI $>30kg/m^2$
- Previous delivery of a baby over 4.5kg - which qualifies this patient
- Previous gestational diabetes
- Family history of diabetes (1st degree relative)
- Minority ethnic family origin with a high prevalence of diabetes

If any one of these risk factors is present then one should offer testing for gestational diabetes. The gold standard testing for patients with risk factors is 2-hour 75g oral glucose tolerance test (OGTT) at 24-28 weeks gestation. If the patient has had gestational diabetes in a previous pregnancy then early-self monitoring of blood glucose or OGTT as soon as possible after booking could also be used for diagnosis.

A diagnosis of gestational diabetes is made if the patient has either:
- Fasting glucose of 5.6 mmol/L or above OR
- A 2-h plasma glucose of 7.8 mmol/L or above

👍 👎 💬 Discuss (8) Improve

Next question

Gestational diabetes ★

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:
- 87.5% have gestational diabetes
- 7.5% have type 1 diabetes
- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes
- BMI of > 30 kg/m^2
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes

- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)
- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:
- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose
- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
    - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
    - if glucose targets are still not met insulin should be added to diet/exercise/metformin
    - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios,

insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets with metformin but decline insulin treatment

Management of pre-existing diabetes

- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	**Target**
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

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Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

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1 ✗

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You are called to urgently review a 45-year-old woman in the emergency department. She presented earlier in the day with fever, a productive cough and confusion. She had been diagnosed by the emergency department doctors as having community acquired pneumonia after seeing consolidation on her chest X-ray and sepsis secondary to this with associated delirium. Collateral history from her partner establishes that she had been feeling unwell for two days with a cough and become increasingly unwell, confused and agitated. The partner also explained that she had recently been diagnosed with an overactive thyroid and been told to start urgently on anti-thyroid tablets, but had not done so when she had read the label of these tablets and found that they should not be taken during pregnancy and she is trying to become pregnant. She had been taking paracetamol only for fever.

Despite treating with IV co-amoxiclav and oral clarithromycin and fluid resuscitation with three liters of fluid as well as IV paracetamol the patient has not improved.

On examination she is very agitated and aggressive and is difficult to examine. She feels very hot and clammy to touch, but peripheries are well perfused. Auscultation shows left sided crepitations. Her heart rate is 170 bpm, blood pressure 81/40 mmHg, temperature 39.2°C, oxygen saturations 100% on 2L via nasal cannulae.

Blood tests from this admission are still pending. Out-patient blood tests show a undetectable low TSH two weeks ago, as well as a free T4 of 53mmol/ml.

What is the most appropriate immediate treatment?

○ Change to IV meropenem and monitor ×

○ Start carbimazole ×

○ Urgent referral for thyroidectomy ×

○ Start IV propranolol ×

○ IV adenosine ×

Submit answer

Reference ranges ∨

Score: **0%**

1 ×
2 -

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Blood tests from this admission are still pending. Out-patient blood tests show a undetectable low TSH two weeks ago, as well as a free T4 of 53mmol/ml.

What is the most appropriate immediate treatment?

Change to IV meropenem and monitor	1%
Start carbimazole	11%

Urgent referral for thyroidectomy	2%
Start IV propranolol	84%
IV adenosine	1%

In thyroid storm with IV beta-blockers are a important first-line treatment

Important for me Less important

This is a patient with severe confusion, fever and evidence of chest infection and untreated hyperthyroidism. The clinical history suggests that she has developed a thyroid storm as she has fever, severe tachycardia, confusion and agitation with hyperthyroidism. This is a medical emergency, and the most urgent matter is to control her heart rate which is likely the cause for her low blood pressure. IV beta-blocker should be started for this purpose, and propranolol would further help reduce the conversion of T3 into T4. In addition, fluids and dexamethasone should be given, as well as anti-thyroid medications.

Changing to meropenem is unlikely to add any further benefit as the effects of the co-amoxiclav probably have not been seen yet. As the patient is not stable it is not appropriate to refer for surgery as management, but may be a viable long-term strategy to treat her hyperthyroidism. Starting carbimazole now is not the most appropriate action as the patient is unstable and this needs to be corrected first. Adenosine is would be appropriate for a patient with SVT without infection and thyrotoxicosis.

Discuss (13) Improve

Next question

Thyroid storm

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:

- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:

- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:

- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

Next question

Textbooks

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Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

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2 ✗

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A 62-year-old woman with a history of type 2 diabetes comes to the clinic for review. She has a history of mild cardiac failure managed with ramipril and bisoprolol. Her current medication for diabetes is metformin 1g BD. On examination her blood pressure is 122/82 mmHg, pulse is 80 beats per minute and regular. There are bilateral basal crackles on auscultation of the chest, and pitting oedema of both ankles. Her body mass index is elevated at 33 kg/m².

HbA1c	73 mmol/mol
Creatinine	82 µmol/l

Which of the following is the most appropriate next step for managing glucose control?

- ○ Empagliflozin ✕
- ○ Glipizide ✕
- ○ Liraglutide ✕
- ○ Pioglitazone ✕
- ○ Saxagliptin ✕

Submit answer

Reference ranges

Score: **0%**

1	✗
2	✗
3	-

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← Question 3 of 217 ✕ ⚐ →

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HbA1c	73 mmol/mol
Creatinine	82 µmol/l

Which of the following is the most appropriate next step for managing glucose control?

🟩	Empagliflozin	90%
	Glipizide	2%
	Liraglutide	5%
	Pioglitazone	1%
🟥	Saxagliptin	2%

This patient has moderate obesity with a BMI of 33, cardiac failure, and a poorly controlled HbA1c at 33. Out of the available options empagliflozin, an SGLT-2 inhibitor is the most appropriate. As well as lowering blood glucose, SGLT-2 inhibition also has a diuretic like effect, lowering blood pressure and reducing fluid retention. Empagliflozin has also been shown to reduce cardiovascular mortality, and this patient is at high risk of an event.

Glipizide is inappropriate as it may drive further weight gain in a patient who is already obese, saxagliptin is recognised to cause heart failure, and pioglitazone has also been shown in clinical trials to cause fluid retention. This leaves liraglutide as the other potential option, which doesn't significantly impact on fluid balance and according to NICE is only indicated at BMI of 35 and above.

👍 👎 Discuss (4) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

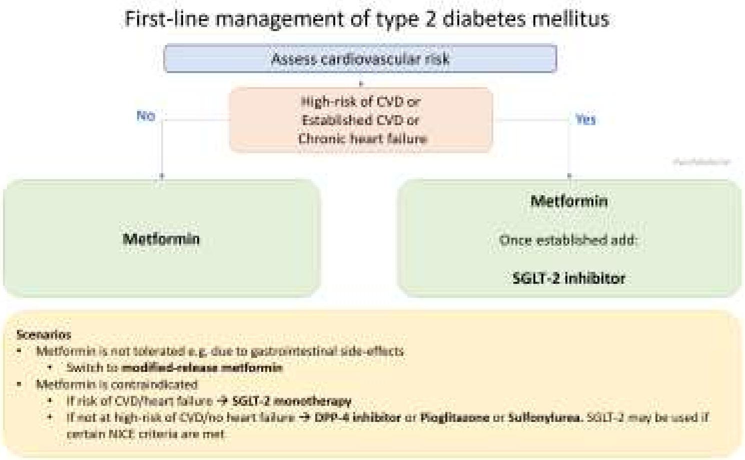
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios

- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

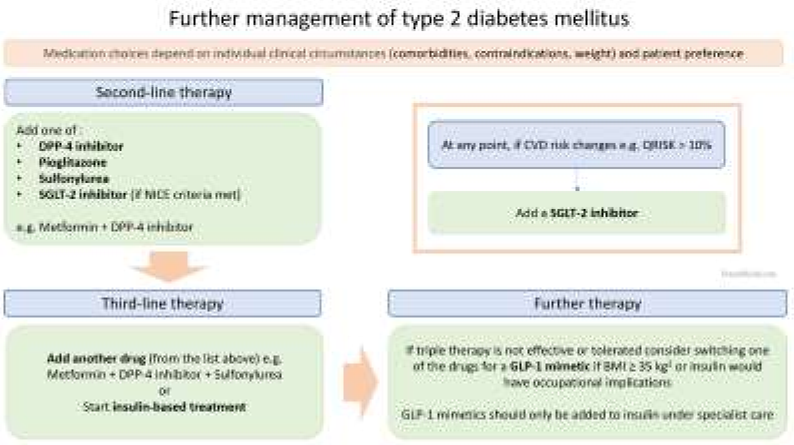
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a

QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated
- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone

- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

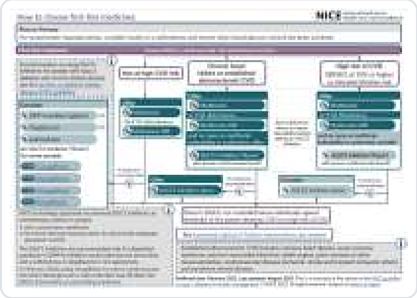
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

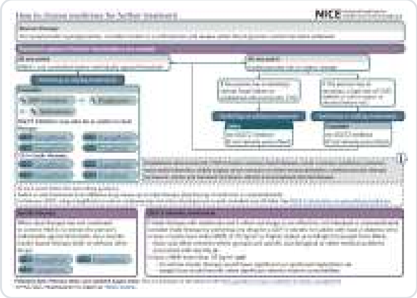
GLP-1 mimetics should only be added to insulin under specialist care

Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

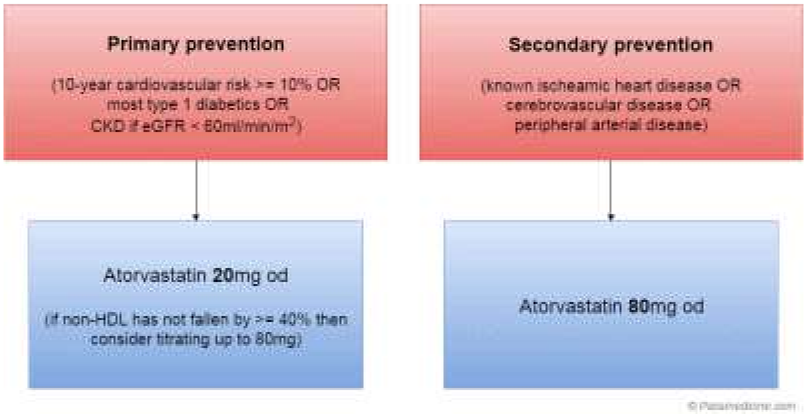
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischaemic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

+ Q 123

Next question

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Links

NICE 33 40

[2014 Lipid modification guidelines]

SIGN 36 41

[2010 Management of diabetes]

NICE 👍 56 👎 48

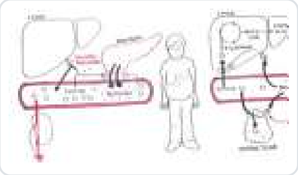
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

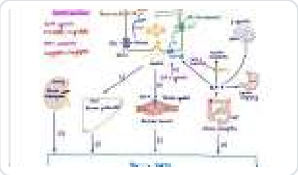
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

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1	✗
2	✗
3	✗

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Question 4 of 217

A 32-year-old pregnant woman (26 weeks) presents to her GP because she has been feeling increasingly tired. She is normally fit and well and has had one previous pregnancy which was uneventful. A fasting blood glucose is 5.4 mmol/L. Urine dip is negative and her blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Blood results are as follows:

Hb	116 g/L	Male: (135-180) Female: (115 - 160)
Platelets	270 * 10^9/L	(150 - 400)
WBC	7.3 * 10^9/L	(4.0 - 11.0)

Na^+	136 mmol/L	(135 - 145)
K^+	3.9 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)

Bilirubin	4 µmol/L	(3 - 17)
ALP	170 u/L	(30 - 100)
ALT	20 u/L	(3 - 40)
Î³GT	55 u/L	(8 - 60)
Albumin	37 g/L	(35 - 50)

Which of the following is the most likely diagnosis?

○ Acute cholecystitis ✕

- ○ Gestational diabetes ✕
- ○ Intrahepatic cholestasis of pregnancy ✕
- ○ Non-alcoholic fatty liver disease ✕
- ○ Normal pregnancy ✕

Submit answer

Reference ranges

Score: **0%**

1 ✕
2 ✕
3 ✕
4 -

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○ Gestational diabetes ✕

○ Intrahepatic cholestasis of pregnancy ✕

○ Non-alcoholic fatty liver disease ✕

○ Normal pregnancy ✕

Submit answer

Reference ranges ⌄

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2 ✕
3 ✕
4 -

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ALT	20 u/L	(3 - 40)
Î³GT	55 u/L	(8 - 60)
Albumin	37 g/L	(35 - 50)

Which of the following is the most likely diagnosis?

Acute cholecystitis	1%

Gestational diabetes	6%
Intrahepatic cholestasis of pregnancy	18%
Non-alcoholic fatty liver disease	2%
Normal pregnancy	73%

Normal pregnancy can cause a raised ALP - it doesn't necessarily imply liver problems

Important for me Less important

The only abnormal finding is a slightly raised ALP. ALP is found in bone, liver, kidney, and intestinal and placental tissue, so ALP tends to rise during pregnancy. In pregnant women levels of up to twice that upper limit can still be normal.

Acute cholecystitis would usually present with abdominal symptoms and right upper quadrant tenderness. It is important to bear in mind that the risk of gallstones increases during pregnancy, as oestrogen can lead to higher cholesterol levels in bile.

Diagnosis (according to NICE) can be made based on either a fasting plasma glucose level of 5.6 mmol/L or above, or a 2-hour plasma glucose level of 7.8 mmol/L or above. Her fasting plasma glucose is below the threshold.

Intrahepatic cholestasis of pregnancy typically presents with itching (particularly in palms and soles) and can lead to complications for both mother and baby if untreated. LFTs are usually deranged but may be normal on some occasions.

Non-alcoholic fatty liver disease (NAFLD) is excessive fat build-up in the liver without another clear cause. NAFLD can progress to non-alcoholic steatohepatitis (NASH). Elevated liver enzymes are common in both but would not typically present with just an isolated rise in ALP. There is also no reason for this young fit woman to have NAFLD.

Discuss (3) Improve

Next question

Alkaline phosphatase

Causes of raised alkaline phosphatase (ALP)

- liver: cholestasis, hepatitis, fatty liver, neoplasia
- Paget's
- osteomalacia
- bone metastases
- hyperparathyroidism
- renal failure
- physiological: pregnancy, growing children, healing fractures

The table below splits the causes according to the calcium level

Raised ALP and raised calcium	Raised ALP and low calcium
Bone metastases Hyperparathyroidism	Osteomalacia Renal failure

+ Q 123

Next question

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1	✗
2	✗
3	✗
4	✓

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Question 5 of 217

A 28-year-old woman presents with flu-like symptoms, palpitations and pain over the anterior neck over the past 2-3 weeks. She has also suffered rapid weight loss and feels increasingly anxious that there may be something seriously wrong with her. Her thyroid-stimulating hormone has been measured at <0.05 IU by her GP. On examination her blood pressure is 128/82 mmHg, her pulse is 95 beats per minute and regular, and she has a fine tremor. There is mild tenderness over the anterior neck. Body mass index is 22 kg/m²

Which of the following would you also expect to find?

○ Erythema nodosum ×

○ Exophthalmos ×

○ Multiple small thyroid nodules on ultrasound scan ×

○ Positive anti-thyroid antibodies ×

○ Reduced uptake on thyroid scintigraphy ×

Submit answer

Reference ranges

Score: **25%**

1 ×

2 ×

3	✗
4	✓
5	-

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Which of the following would you also expect to find?

	Erythema nodosum	2%
	Exophthalmos	5%
	Multiple small thyroid nodules on ultrasound scan	3%
🟥	Positive anti-thyroid antibodies	9%
🟩	Reduced uptake on thyroid scintigraphy	80%

In De Quervain's thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy

Important for me Less important

The most likely diagnosis, given the history of 2-3 weeks of flu-like symptoms and suppressed TSH, is subacute thyroiditis where thyroid inflammation drives increased release of stored thyroid hormone, rather than the clinical picture being due to overproduction of T3 and T4. Symptoms of hyperthyroidism should be managed with beta blockade as required, and there is no role for thioamides. Pain over the thyroid can be managed with non-steroidal anti-inflammatory drugs. After a period of hyperthyroidism, rebound hypothyroidism may be seen, followed by a recovery to euthyroidism.

Erythema nodosum is not associated with subacute thyroiditis. Exophthalmos and positive anti-thyroid antibodies are associated with autoimmune thyroid disease. Multiple small thyroid nodules are a feature of multinodular goitre.

👍 👎 Discuss (4) Improve

Next question

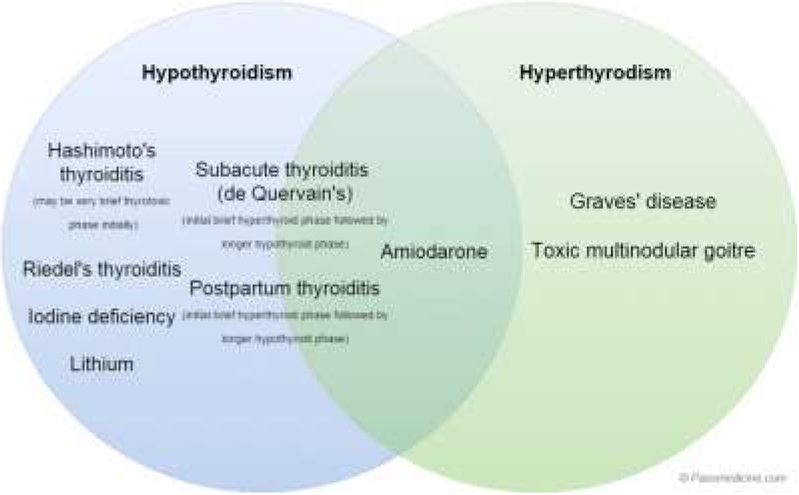
Subacute (De Quervain's) thyroiditis ★

Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations
- thyroid scintigraphy: globally reduced uptake of iodine-131

Management
- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

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1 ✗
2 ✗
3 ✗
4 ✓
5 ✗

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Question 6 of 217

A 45-year-old woman with chronic alcohol abuse admitted 3 days ago for nausea and severe diarrhoea now complains of peri-oral and finger tingling. She was admitted for hydration after 1 week of severe watery diarrhoea. She has been receiving intravenous hydration and dextrose but has not been able to take oral nutrition secondary to continued nausea. Her blood pressure is 130/74 mmHg, pulse is 68/min, and respiratory rate is 16/min. She is afebrile.

Physical examination is significant for facial twitching on percussion of her facial nerve just anterior to the ear, as well as the induction of carpal spasm after the inflation of a blood pressure cuff on her arm.

Which of the following is most likely to have caused these findings?

- ○ Hyperuricaemia ×
- ○ Hypernatraemia ×
- ○ Hypomagnesaemia ×
- ○ Hypophosphataemia ×
- ○ Hypouricaemia ×

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Reference ranges

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1	✗
2	✗
3	✗
4	✓
5	✗
6	-

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Physical examination is significant for facial twitching on percussion of her facial nerve just anterior to the ear, as well as the induction of carpal spasm after the inflation of a blood pressure cuff on her arm.

Which of the following is most likely to have caused these findings?

Hyperuricaemia	2%
Hypernatraemia	0%
Hypomagnesaemia	70%
Hypophosphataemia	25%
Hypouricaemia	1%

Magnesium deficiency causes hypocalcaemia

Important for me Less important

This patient is displaying classic signs of hypocalcaemia, including hyperexcitability of her facial nerve (Chvostek's sign), induced carpal spasm (Trousseau's sign), and tingling of the extremities and lips. Calcium homeostasis is a complicated process involving PTH, vitamin D, albumin and numerous electrolytes. Acquired hypoparathyroidism is the most common form of true hypocalcaemia, most often occurring transiently after thyroid surgery or after the removal of a parathyroid adenoma. Occasionally, hypomagnesaemia can produce hypocalcaemia by decreasing both the body's production of PTH and its sensitivity to the hormone. In this case, it is likely that the patient became magnesium depleted from her course of watery diarrhoea, likely baseline poor nutritional status and alcohol abuse.

Choice 1: Hyperuricaemia is not a cause of hypocalcaemia. Chronic kidney disease, however can lead to hypocalcaemia in the setting of secondary hyperparathyroidism, but there is no evidence of renal failure in this patient.

Choice 2: Fluid balance (hyper- or hyponatraemia) does not play a role in calcium homeostasis.

Choice 4: Hypophosphataemia is not a cause of hypocalcaemia. Actually, hypocalcaemia often leads to hyperphosphataemia secondary to increased PTH-mediated bone resorption. Elevations in phosphate may also contribute to hypocalcaemia by complexing with circulating calcium and suppressing conversion of 25-OH to 1, 25-OH vitamin D.

Choice 5: Urate levels do not affect calcium homeostasis.

👍 👎 Discuss (1) Improve

Next question

Hypocalcaemia: causes and management ★

The clinical history combined with parathyroid hormone levels will reveal the cause of hypocalcaemia in the majority of cases

Causes
- vitamin D deficiency (osteomalacia)
- chronic kidney disease
- hypoparathyroidism (e.g. post thyroid/parathyroid surgery)
- pseudohypoparathyroidism (target cells insensitive to PTH)
- rhabdomyolysis (initial stages)

- magnesium deficiency (due to end organ PTH resistance)
- massive blood transfusion
- acute pancreatitis

Contamination of blood samples with EDTA may also give falsely low calcium levels.

Management
- severe hypocalcaemia (e.g. carpopedal spasm, tetany, seizures or prolonged QT interval) requires IV calcium replacement
    - the preferred method is with intravenous calcium gluconate, 10ml of 10% solution over 10 minutes
    - intravenous calcium chloride is more likely to cause local irritation
    - ECG monitoring is recommended
- further management depends on the underlying cause

+ Q 123

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Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 3 👎 2

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1	✗
2	✗
3	✗
4	✓
5	✗
6	✓

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Question 7 of 217

A 75-year-old man presents to referred to the diabetic clinic by his general practitioner with newly identified hyperglycaemia. He had presented with a two month history of polyuria, polydipsia and diarrhoea and was found to have a blood sugar of 18.4 mmol/L. Over this time period he had lost 6kg and now weighed 61kg. His past medical history includes hypertension and a deep vein thrombosis which was diagnosed three months ago. He takes amlodipine 10mg and warfarin.

What is the diagnosis?

- ○ Glucagonoma ×
- ○ Type one diabetes mellitus ×
- ○ Cushing's syndrome ×
- ○ Drug induced diabetes ×
- ○ Type two diabetes mellitus ×

Submit answer

Reference ranges

Score: **33.3%**

1 ✗
2 ✗
3 ✗

4	✓
5	✗
6	✓
7	-

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What is the diagnosis?

🟩	Glucagonoma	63%
	Type one diabetes mellitus	3%
	Cushing's syndrome	5%
	Drug induced diabetes	6%
🟥	Type two diabetes mellitus	24%

Glucagonoma is an uncommon tumour of the pancreatic alpha cells. It can present with new or worsening diabetes mellitus, venous thromboembolism, the classic rash of necrolytic migratory erythema (a painful, pruritic maculopapular rash occuring typically at sites of friction with clothing...) and other symptoms of hyperglucagonaemia (diarrhoea, weight loss, anaemia). Type one diabetes mellitus can of course present with the osmotic symptoms of hyperglycaemia and weight loss but would be unlikely in a patient of this age with no history of auto-immune disease. Type two diabetes mellitus would be uncommon in a non-obese older patient and is less likely to present with osmotic symptoms.

👍 | 👎 | Discuss (5) | Improve

Next question

Glucagonoma

Glucagonomas are small tumours, almost always found in the pancreas, and frequently malignant. The tumours arise from the alpha cells of the pancreas.

They present with diabetes mellitus, venous thrombo-embolism and the classical rash of necrolytic migratory erythema - a red, blistering rash.

A serum level of glucagon >1000pg/ml usually suggests the diagnosis, imaging with CT scanning is also required.

Treatment options include surgical resection and octreotide.

+ Q 123

Next question

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Score: **28.6%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗

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A 78-year-old man is admitted with severe diarrhoea. He states that he has had profuse diarrhoea for the past 6 days. He denies blood or mucus in his stools. On examination he is peripherally shut down. His blood pressure is 85/45 mmHg and heart rate is 125 beats per minute. Blood results are as follows:

Hb	128 g/l	Na$^+$	112 mmol/l
Platelets	488 * 10^9/l	K$^+$	3.2 mmol/l
WBC	14.8 * 10^9/l	Urea	18.8 mmol/l
Neuts	10.8 * 10^9/l	Creatinine	212 µmol/l
Lymphs	2.2 * 10^9/l	CRP	78 mg/l

You diagnose hypovolaemic shock and resuscitate him with 0.9% saline boluses.

Later on in the day the patient presents with muscle weakness. On examination you note spastic quadriparesis.

What is the most likely cause?

- ○ Cerebral oedema ×
- ○ Central pontine myelinolysis (CPM) ×
- ○ Cerebral vasospasm ×
- ○ Cerebral hypoxia secondary to prolonged hypotension ×
- ○ Brain stem infarct ×

Submit answer

Reference ranges

Score: **28.6%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	-

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Question 8 of 217 ✓

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Later on in the day the patient presents with muscle weakness. On examination you note spastic quadriparesis.

What is the most likely cause?

Cerebral oedema	9%
[✓ Central pontine myelinolysis (CPM)]	88%
Cerebral vasospasm	1%
Cerebral hypoxia secondary to prolonged hypotension	2%
Brain stem infarct	0%

Rapid correction of hyponatraemia can result in central pontine myelinolysis (CPM). CPM features are variable, however the classical presentation is spastic quadriparesis, pseudobulbar palsy, and emotional lability (pseudobulbar affect)

Important for me Less important

The patient has marked hypovolaemic hyponatraemia secondary to profuse diarrhoea. Large volumes of saline has resulted in rapid correction of the hyponatraemia. Sudden rises in plasma osmolarity can result in fluid shift from the cerebral intracellular space to the extravascular space resulting in cerebral shrinking and demyelination. This end result is central pontine myelinolysis (CPM).

CPM features are variable, however the classical presentation is spastic quadriparesis, pseudobulbar palsy, and emotional lability (pseudobulbar affect).

👍 👎 Discuss (11) Improve

Next question

Hyponatraemia: treatment ★

Untreated, severe hyponatraemia may result in cerebral oedema, which in turn can cause brain herniation. It is therefore important to promptly identify and treat hyponatremia appropriately. However, the particular management for each patient is based on a number of factors listed below. Further to this, over-rapid correction may result in osmotic demyelination syndrome.

Principles

Management of hyponatremia is complicated and primarily based on the following parameters:

- duration of hyponatremia: is it acute or chronic?
    - acute: develops over a period of < 48 hours
    - chronic: develops over a period > 48 hours
- the severity of hyponatremia: what is the sodium level?
    - mild: 130-134 mmol/L

    - moderate: 120-129 mmol/L
    - severe: < 120 mmol/L
- symptoms: is the patient symptomatic?
    - patients with mild hyponatraemia may be symptomatic
    - early symptoms may include: headache, lethargy, nausea, vomiting, dizziness, confusion, and muscle cramps
    - late symptoms may include: seizures, coma, and respiratory arrest
- the suspected aetiology of the hyponatraemia:
    - hypovolemic hyponatraemia/clinically dehydrated: diuretic stage of renal failure, diuretics, Addisonian crisis
    - euvolemic hyponatraemia: SIADH
    - hypervolaemic hyponatraemia: heart failure, liver failure, nephrotic syndrome

Management

Initial steps in all patients

- exclude a spurious result (e.g. blood taken from a drip arm)
- review medications that may cause hyponatraemia

Chronic hyponatreamia without severe symptoms

If a hypovolemic cause is suspected

- normal, i.e. isotonic, saline (0.9% NaCl)
- this may sometimes be given as a trial
    - if the serum sodium rises this supports a diagnosis of hypovolemic hyponatraemia
    - if the serum sodium falls an alternative diagnosis such as SIADH is likely

If a euvolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider medications:
    - demeclocycline
    - vaptans (see below)

If a hypervolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider loop diuretics
- consider vaptans

Acute hyponatreamia with severe symptoms

Patients with acute, severe (<120 mmol/L) or symptomatic hyponatraemia require close monitoring, preferably in an HDU or above setting.

Hypertonic saline (typically 3% NaCl) is used to correct the sodium level more quickly than would be done in patients with chronic hyponatraemia.

Notes on specific medications

Vasopressin/ADH receptor antagonists (vaptans):

- these act primarily on V2 receptors - antagonism of V2 receptors results in selective water diuresis, sparing the electrolytes
- They should be avoided in patients who have hypovolemic hyponatremia
- Vasopression/ADH receptor antagonists can stimulate the **thirst** receptors leading to the desire to drink free water. They can be **hepatotoxic** in patients with underlying liver disease.

Complications of treatment

Osmotic demyelination syndrome (central pontine myelinolysis)

- can occur due to over-correction of severe hyponatremia
- pathophysiology:
    - thought to develop secondary to astrocyte (and possibly oligodendrocyte) apoptosis
    - astrocytes and oligodendrocytes (cells of the glial syncytium) are crucial for normal myelination

    - chronic hyponatraemia → loss of osmotically active organic osmolytes (such as myoinositol, glutamate, glutamine) from astrocytes. These provide protection against cerebral oedema
    - organic osmolytes cannot be replaced quickly enough when the brain volume begins to shrink in response to the correction of hyponatraemia
    - the dehydrated astrocytes and oligodendrocytes undergo apoptosis or other forms of injury → demyelination
- to avoid this, Na+ levels are only raised by 4 to 6 mmol/l in a 24-hour period
- symptoms usually occur after 2 days and are usually irreversible: dysarthria, dysphagia, paraparesis or quadriparesis, seizures, confusion, and coma
- patients are awake but are unable to move or verbally communicate, also called '**Locked-in syndrome**'

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 24 28

2017 Hyponatraemia â€“ presentations and management

Suggest link Report broken link

Score: **37.5%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓

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A 42-year-old woman was seen in clinic with a history of palpitation, tremor and weight loss. There is no other past medical history and she takes no regular medication.

On examination, she had a palpable goitre, exophthalmos, and a tremor of the out-stretched hands.

Thyroid function tests showed:

Free thyroxine (T4)	36 pmol/L (10-25)
Free triiodothyronine (T3)	15 pmol/L (5-10)
Thyroid-stimulating hormone	0.1mU/L (0.4-5.0)

Which of the following treatments should be prescribed initially to improve symptoms?

- ○ Thyroidectomy ×
- ○ Propanolol ×
- ○ Radioiodine ablation ×
- ○ Carbimazole ×
- ○ Propylthiouracil ×

Submit answer

Reference ranges

Score: **37.5%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	-

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Question 9 of 217 ✓

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Thyroid-stimulating hormone	0.1mU/L (0.4-5.0)

Which of the following treatments should be prescribed initially to improve symptoms?

Thyroidectomy	1%
Propanolol	74%
Radioiodine ablation	1%
Carbimazole	20%
Propylthiouracil	5%

Propranolol should be used in new cases of Graves' disease to help control symptoms

Important for me Less important

There is a high suspicion of Grave's disease in this patient, and further investigation with a thyroid autoantibody profile is warranted.

Beta blockers are used to treat the symptoms of increased beta-adrenergic tone that are seen in Grave's disease. Other such symptoms include anxiety and heat intolerance.

Thioamides are used to treat Grave's hyperthyroidism, but not specifically the symptoms of increased beta-adrenergic tone. While the anti-thyroid effect of these drugs has a rapid onset, their clinical effect is more delayed because the pre-formed store of hormone in the thyroid gland and bound to thyroid-binding globulin must first be exhausted.

👍 👎 Discuss (6) Improve

Next question

Graves' disease: management

Despite many trials, there is no clear guidance on the optimal management of Graves' disease. Treatment options include anti-thyroid drugs (ATDs, for example carbimazole), radioiodine treatment and surgery.

Anti-thyroid drugs have emerged as the most popular first-line therapy for Graves' disease in recent years. Factors that particularly support their use include anti-thyroid drugs significant symptoms of thyrotoxicosis, or patients with a significant risk of hyperthyroid complications (e.g. elderly patients, cardiovascular disease).

Initial treatment to control symptoms

- propranolol is used to help block the adrenergic effects

NICE recommends that patients with Graves' disease are referred to secondary care for ongoing treatment.

- NICE suggest carbimazole should be considered in primary care if patients symptoms are not controlled with propanolol

ATD therapy

- carbimazole is started at 40mg and reduced gradually to maintain euthyroidism
- typically continued for 12-18 months
- the major complication of carbimazole therapy is agranulocytosis
- an alternative regime is termed 'block-and-replace'
    - carbimazole is started at 40mg
    - thyroxine is added when the patient is euthyroid
    - treatment typically lasts for 6-9 months
    - patients following an ATD titration regime have been shown to suffer fewer side-effects than those on a block-and-replace regime

Radioiodine treatment

- often used in patients who relapse following ATD therapy or are resistant to primary ATD treatment
- contraindications include pregnancy (should be avoided for 4-6 months following treatment) and age < 16 years. Thyroid eye disease is a relative contraindication, as it may worsen the condition
- the proportion of patients who become hypothyroid depends on the dose given, but as a rule the majority of patient will require thyroxine supplementation after 5 years

+ Q 123

Next question

Textbooks

Dr. Jamil Alrajhi

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 11 👎 4

Graves' disease: management

Suggest link

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Media

HYPERTHYROIDISM
TREATMENT MEDICATIONS

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 0

ENDOCRINOLOGY
GRAVES DISEASE

Graves' disease

Armando Hasudungan - YouTube 👍 2 👎 2

Report broken media

Score: **44.4%**

1 ✕

2 ✕

3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓

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A 43-year-old woman attends with a 1-year history of recurrent lethargy, shakiness, slurred speech, and diplopia. Symptoms most commonly develop in the mornings and then resolve after breakfast.

She has a past medical history of type 2 diabetes mellitus, alcohol excess and a functional neurological disorder.

Observations: SpO2 97% on room air, respiratory rate 22 breaths/minute, blood pressure 92/64, heart rate 116 beats per minute, apyrexial

Capillary blood glucose: 5.2

During your review, she becomes shaky and feels unwell. A repeat blood glucose is 2.4.

You send a set of blood tests and then administer 40% dextrose gel. The blood tests are reported as follows:

Insulin	43 pmol/L	(<25)
C-peptide	114 pmol/L	(<75)
Pro-insulin	23 pmol/L	(3.6-22)

What is the most likely diagnosis?

- ○ Alcohol-related hypoglycaemia ×
- ○ Exogenous insulin administration ×
- ○ Glimepiride use ×
- ○ Insulinoma ×
- ○ Non-insulinoma pancreatogenous hypoglycaemia syndrome ×

Submit answer

Reference ranges ⌄

Score: **44.4%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	-

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C-peptide	114 pmol/L	(<75)
Pro-insulin	23 pmol/L	(3.6-22)

What is the most likely diagnosis?

Alcohol-related hypoglycaemia	2%
Exogenous insulin administration	9%
Glimepiride use	17%
Insulinoma	67%
Non-insulinoma pancreatogenous hypoglycaemia syndrome	4%

An elevated C-peptide level following administration of IV insulin can confirm insulinoma

Important for me Less important

The most likely diagnosis here is insulinoma. This should be suspected in patients with symptoms of hypoglycaemia that improve after eating. Symptoms of hypoglycaemia can be varied and diagnosis of insulinoma can take months to years due to difficult diagnosis.

Insulinoma is correct. An insulinoma is a rare neuroendocrine tumour that secretes insulin, as a result insulin, pro-insulin, and C-peptide levels are raised.

Exogenous insulin administration is incorrect. Pro-insulin cleavage results in C-peptide and insulin which are then released from pancreatic beta cells. It is a marker of insulin secretion. C-peptide does not form part of exogenous insulin injections and so would not be raised if insulin was being administered erroneously.

Glimepiride use is incorrect. Glimepiride is a sulfonylurea and so can be associated with episodes of hypoglycaemia. It can cause a rise in insulin, C-peptide, and pro-insulin levels as seen here, however insulin levels would generally not be increased to this degree. To confirm or refute the diagnosis a supervised fast may be appropriate.

Alcohol-related hypoglycaemia is incorrect. Alcohol causes hypoglycaemia by inhibiting gluconeogenesis. In these cases, you would expect to see low levels of insulin and C-peptide in response to low blood glucose levels.

Non-insulinoma pancreatogenous hypoglycaemia syndrome is incorrect. This is a rare syndrome characterised by post-prandial hypoglycaemia episodes and a normal 72-hour fast. This is usually due to a genetic mutation and pancreatic hyperplasia. You may expect to see elevated insulin, pro-insulin, and C-peptide, but the differentiating factor here is that the hypoglycaemic episodes commonly occur after meals which would be unusual in insulinoma.

Discuss (13) Improve

Next question

Insulinoma

An insulinoma is a neuroendocrine tumour deriving mainly from pancreatic Islets of Langerhans cells

Basics

- most common pancreatic endocrine tumour
- 10% malignant, 10% multiple
- of patients with multiple tumours, 50% have MEN-1

Features

- of hypoglycaemia: typically early in morning or just before meal, e.g. diplopia, weakness etc
- rapid weight gain may be seen
- high insulin, raised proinsulin:insulin ratio
- high C-peptide

Diagnosis

- supervised, prolonged fasting (up to 72 hours)
- CT pancreas

Management

- surgery
- diazoxide and somatostatin if patients are not candidates for surgery

+ Q 123

Next question

Textbooks

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Media

PANCREATIC
NEUROENDOCRINE
NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **50%**

1 ✗
2 ✗
3 ✗
4 ✓
5 ✗
6 ✓
7 ✗

8	✓
9	✓
10	✓

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Question 11 of 217

A 19-year-old woman comes to the endocrine clinic for review. She has problems with hirsutism and irregular periods, and troublesome weight gain. Her GP has just stressed the need to lose weight and offered no pharmacological intervention. She takes no medication from the doctor and is currently studying law. Examination reveals a blood pressure of 135/85 mmHg, pulse is 65 beats per minute and regular. body mass index is 32kg/m². You confirm extensive hirsutism affecting the beard line, upper lip and the nipples. there is acne over the face and the upper chest. Relevant bloods include:

testosterone	4.8 nmol/l (upper limit of normal 2.1 nmol/l)
LH:FSH ratio	2.1
fasting glucose	5.0 mmol/l

Her main concern is hirsutism.

Which of the following is the most appropriate intervention?

- ○ Co-cyprindiol ✕
- ○ Clomiphene ✕
- ○ Levonorgestrel ✕
- ○ Metformin ✕
- ○ Pioglitazone ✕

Submit answer

Reference ranges

Score: **50%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	-

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Question 11 of 217 ✗

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LH:FSH ratio	2.1
fasting glucose	5.0 mmol/l

Her main concern is hirsutism.

Which of the following is the most appropriate intervention?

🟩	Co-cyprindiol	56%
🟥	Clomiphene	16%
	Levonorgestrel	10%
	Metformin	18%
	Pioglitazone	0%

Co-cyprindiol contains both cyproterone, an anti-androgen, and ethinylestradiol, (a synthetic oestrogen). In combination, used for the treatment of polycystic ovarian syndrome, the most likely diagnosis here, co-cyprindiol significantly reduces symptoms of hirsutism and acne, both related to androgen excess.

Clomiphene is the preferred option for inducing ovulation, and is preferred to metformin for this purpose, although the two are sometimes used in combination in the obese population. Pioglitazone is also effective in reducing ovarian insulin resistance, and inducing ovulation, but is not used due to its adverse event profile. Progesterone, (levonorgestrel), is ineffective in managing hirsutism.

👍 👎 Discuss (4) Improve

Next question

Polycystic ovarian syndrome: management ★

Polycystic ovarian syndrome (PCOS) is a complex condition of ovarian dysfunction thought to affect between 5-20% of women of reproductive age. Management is complicated and problem based partly because the aetiology of PCOS is not fully understood. Both hyperinsulinaemia and high levels of luteinizing hormone are seen in PCOS and there appears to be some overlap with the metabolic syndrome.

General

- weight reduction if appropriate
- if a women requires contraception then a combined oral contraceptive (COC) pill may help regulate her cycle and induce a monthly bleed (see below)

Hirsutism and acne

- a COC pill may be used help manage hirsutism. Possible options include a third generation COC which has fewer androgenic effects or co-cyprindiol which has an anti-androgen action. Both of these types of COC may carry an increased risk of venous thromboembolism
- if doesn't respond to COC then topical eflornithine may be tried
- spironolactone, flutamide and finasteride may be used under specialist supervision

Infertility

- weight reduction if appropriate
- the management of infertility in patients with PCOS should be supervised by a specialist. There is an ongoing debate as to whether metformin, clomifene or a combination should be used to stimulate ovulation
- a 2007 trial published in the New England Journal of Medicine suggested clomifene was the most effective treatment. There is a potential risk of multiple pregnancies with anti-oestrogen* therapies such as clomifene. The RCOG published an opinion paper in 2008 and concluded that on current evidence metformin is not a first line treatment of choice in the management of PCOS
- metformin is also used, either combined with clomifene or alone, particularly in patients who are obese
- gonadotrophins

*work by occupying hypothalamic oestrogen receptors without activating them. This interferes with the binding of oestradiol and thus prevents negative feedback inhibition of FSH secretion

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 1 👎 0

Polycystic ovarian syndrome review

RCOG 👍 0 👎 0

Opinion paper on metformin in PCOS

Royal College of Obstetricians and Gynaecologists 👍 2 👎 1

2014 Long-term Consequences of Polycystic Ovary Syndrome

Clinical Knowledge Summaries 👍 0 👎 1

Polycystic ovarian syndrome

ESHRE (European Society of Human Reproduction and Embryology) 👍 0 👎 1

2018 International Evidence Based Guideline for the Assessment and Management of PCOS

Suggest link

Report broken link

Score: **45.5%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗

8 ✓
9 ✓
10 ✓
11 ✗

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Question 12 of 217

An 89-year-old woman is reviewed in the frailty clinic.

She was recently admitted under the care of the elderly team with an episode of delirium, which was triggered by a variety of factors, including a change in housing and constipation.

A course of trimethoprim was prescribed during this admission for a suspected UTI on the basis of urinary urgency but a subsequent urine specimen showed no growth.

You take a detailed history now that she has recovered from her delirium and you strongly suspect she is suffering from urge incontinence, which significantly impacts her quality of life.

She has previously undergone a period of bladder training for this, but given her symptoms persist, you are keen to start the patient on medication to improve her urge incontinence.

What is the most appropriate medication to use in this case?

- ○ Darifenacin ✕
- ○ Duloxetine ✕
- ○ Mirabegron ✕
- ○ Oxybutynin ✕
- ○ Tolterodine ✕

Submit answer

Reference ranges

Score: **45.5%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	-

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She has previously undergone a period of bladder training for this, but given her symptoms persist, you are keen to start the patient on medication to improve her urge incontinence.

What is the most appropriate medication to use in this case?

	Darifenacin	2%
	Duloxetine	10%
🟩	Mirabegron	63%
🟥	Oxybutynin	17%
	Tolterodine	7%

Anticholinergics for urge incontinence are associated with confusion in elderly people - mirabegron is a preferable alternative

Important for me Less important

Urge incontinence can generally be managed with bladder training, but if this is unsuccessful, a trial of medication is recommended in the NICE guidelines.

The correct answer, in this case, is **mirabegron**. Although anticholinergic drugs are first-line, this is recommended as an acceptable alternative when they are contra-indicated.

Anticholinergic drugs (such as **darifenacin, oxybutynin** and **tolterodine**) are first line, but there are some safety concerns, particularly in elderly patients at risk of delirium. This makes these options incorrect choices for this patient, who has been admitted recently with delirium.

Duloxetine is incorrect. It is not used in the management of urge incontinence but is a second-line treatment for stress incontinence when other measures have failed.

👍 👎 Discuss Improve

Next question

Urinary incontinence ★

Urinary incontinence (UI) is a common problem, affecting around 4-5% of the population. It is more common in elderly females.

Risk factors

- advancing age
- previous pregnancy and childbirth
- high body mass index
- hysterectomy
- family history

Classification

- overactive bladder (OAB)/urge incontinence
    - due to detrusor overactivity

    - the urge to urinate is quickly followed by uncontrollable leakage ranging from a few drops to complete bladder emptying
- stress incontinence: leaking small amounts when coughing or laughing
- mixed incontinence: both urge and stress
- overflow incontinence: due to bladder outlet obstruction, e.g. due to prostate enlargement
- functional incontinence
    - comorbid physical conditions impair the patient's ability to get to a bathroom in time
    - causes include dementia, sedating medication and injury/illness resulting in decreased ambulation

Initial investigation

- bladder diaries should be completed for a minimum of 3 days
- vaginal examination to exclude pelvic organ prolapse and ability to initiate voluntary contraction of pelvic floor muscles ('Kegel' exercises)
- urine dipstick and culture
- urodynamic studies
    - not routinely required before starting initial conservative treatment for urinary incontinence
    - may be considered if the diagnosis is uncertain, mixed incontinence is suspected or surgical intervention is planned

Management depends on whether urge or stress UI is the predominant picture

If urge incontinence is predominant:

- bladder retraining (lasts for a minimum of 6 weeks, the idea is to gradually increase the intervals between voiding)
- bladder stabilising drugs: antimuscarinics are first-line
    - NICE recommend oxybutynin (immediate release), tolterodine (immediate release) or darifenacin (once daily preparation)
    - avoid immediate-release oxybutynin in frail older women
    - solifenacin is an alternative antimuscarinic agent
- mirabegron (a beta-3 agonist) may be useful if there is concern about anticholinergic side-effects in frail elderly patients

- if troublesome nocturia is a feature, desmopressin may be considered
- for women with overactive bladder that has not responded to non-surgical management:
    - if urodynamic investigations determine that detrusor overactivity is causing her overactive bladder symptoms then consider botulinum toxin type A bladder wall injection

If stress incontinence is predominant:

- lifestyle measures, e.g. weight reduction, reducing caffeine intake
- pelvic floor muscle training
    - NICE recommend at least 8 contractions performed 3 times per day for a minimum of 3 months
- surgical procedures include:
    - mid-urethral tape procedures (still recommended by NICE but only after discussing risks and benefits)
    - intramural bulking agent injections
    - colposuspension
- duloxetine may be offered to women if they decline surgical procedures
    - a combined noradrenaline and serotonin reuptake inhibitor
    - mechanism of action: increased synaptic concentration of noradrenaline and serotonin within the pudendal nerve → increased stimulation of urethral striated muscles within the sphincter → enhanced contraction

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 23 👎 30

2019 urinary incontinence guidelines

Suggest link

Report broken link

Score: **41.7%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗

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Question 13 of 217

A 34 year-old woman is seen by her GP for the annual review of her type 1 diabetes. Her most recent HbA1c is 58 mmol/mol and the only problem she has noticed are severe hypoglycaemic episodes during the night around 2-4am, that she noticed she was getting while working night shifts for a local superstore. However, by breakfast time, her blood glucose levels often rise to around 15 mmol/mol. Her insulin regimen is currently a twice daily mixed insulin.

What is the most appropriate change to her current insulin treatment?

- ○ Move to a basal bolus of insulin ×
- ○ Take 1 dextrose tablet at 9pm ×
- ○ Reduce nocturnal insulin dose ×
- ○ Reduce morning insulin dose ×
- ○ Add gliclazide ×

Submit answer

Reference ranges

Score: **41.7%**

1 ✗
2 ✗
3 ✗

4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	-

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Question 13 of 217 ✓

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What is the most appropriate change to her current insulin treatment?

Move to a basal bolus of insulin	50%
Take 1 dextrose tablet at 9pm	1%
Reduce nocturnal insulin dose	44%
Reduce morning insulin dose	4%
Add gliclazide	0%

This patient is experiencing severe nocturnal hypoglycaemic episodes followed by rises in blood glucose by breakfast time. The most appropriate change to the insulin treatment regimen is changing to a basal bolus regime, with one daily injection of background long-acting insulin and three short acting injections of insulin.

Discuss (11) Improve

Next question

Insulin therapy

Insulin therapy revolutionised the management of diabetes mellitus when it was developed in the 1920's. It is still the only available treatment for type 1 diabetes mellitus (T1DM) and is widely used in type 2 diabetes mellitus (T2DM) where oral hypoglycaemic agents fail to gain adequate control.

It can sometimes seem daunting to understand the various types of insulin but it is important you have a basic grasp to avoid potential harm to patients.

Classification of insulin

By manufacturing process

- porcine: extracted and purified from pig pancreas
- human sequence insulin: either produced by enzyme modification of porcine insulin (emp) or biosynthetically by recombinant DNA using bacteria (crb, prb) or yeast (pyr)
- analogues

By duration of action

	Onset	**Peak**	**Duration**
Rapid-acting insulin analogues	5 mins	1 hour	3-5 hours
Short-acting insulin	30 mins	3 hours	6-8 hours
Intermediate-acting insulin	2 hours	5-8 hours	12-18 hours
Long-acting insulin analogues	1-2 hours	Flat profile	Up to 24 hours
Premixed preparations	-	-	-

Patients often require a mixture of preparations (e.g. both short and long acting) to ensure stable glycaemic control throughout the day.

Rapid-acting insulin analogues

- the rapid-acting human insulin analogues act faster and have a shorter duration of action than soluble insulin (see below)
- may be used as the bolus dose in 'basal-bolus' regimes (rapid/short-acting 'bolus' insulin before meals with intermediate/long-acting 'basal' insulin once or twice daily)
- insulin aspart: NovoRapid
- insulin lispro: Humalog

Short-acting insulins
- soluble insulin examples: Actrapid (human, pyr), Humulin S (human, prb)
- may be used as the bolus dose in 'basal-bolus' regimes

Intermidate-acting insulins
- isophane insulin
- many patients use isophane insulin in a premixed formulation with

Long-acting insulins
- insulin determir (Levemir): given once or twice daily
- insulin glargine (Lantus): given once daily

Premixed preparations
- combine intermediate acting insulin with either a rapid-acting insulin analogue or soluble insulin
- Novomix 30: 30% insulin aspart (rapid-acting), 70% insulin aspart protamine (intermediate-acting)
- Humalog Mix25: 25% insulin lispro (rapid-acting), 75% insulin lispro protamine (intermediate-acting); Humalog Mix50: 50% insulin lispro, 50% insulin lispro protamine
- Humulin M3: biphasic isophane insulin (human, prb) - 30% soluble (short-acting), 70% isophane (intermediate-acting)
- Insuman Comb 15: biphasic isophane insulin 9human, prb) - 30% soluble (short-acting), 70% isophane (intermediate-acting)

Administration of insulin

The vast majority of patients administer insulin subcutaneously. It is important to rotate injection sites to prevent lipodystrophy. Insulin pumps are available ('continuous subcutaneous insulin infusions') which delivers a continuous basal infusion and a patient-activated bolus dose at meal times.

Intravenous insulin is used for patients who are acutely unwell, for example with diabetic ketoacidosis. Inhaled insulin is available but not widely used and oral insulin analogues are in development but have considerable technical hurdles to clear.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **46.2%**

1 ✗
2 ✗
3 ✗
4 ✓
5 ✗

6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓

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Back to top

← Question 14 of 217 →

A 32-year-old patient with Addison's disease comes for review into the endocrinology clinic. He was diagnosed by Addison's disease nine months ago and has since then been started on 20mg of hydrocortisone taken in divided doses and 50micrograms of fludrocortisone. He complains of feeling intermittently tired but has otherwise is well. He has a history of hypothyroidism and takes 100 micrograms of levothyroxine for this. He has no other medical problems and feels well in his mood. His most recent TSH was done one month ago, and showed his thyroid function to be well within control. What investigation could be offered to assess if the dose of hydrocortisone represents accurate glucocorticoid replacement?

- ○ Dexamethasone suppression test ×
- ○ Cortisol day curve ×
- ○ Synacthen test ×
- ○ Morning serum cortisol ×
- ○ Urinary electrolytes ×

Submit answer

Reference ranges

Score: **46.2%**

1 ×

2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓
14	-

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	Dexamethasone suppression test	3%
🟩	Cortisol day curve	49%
🟥	Synacthen test	13%
	Morning serum cortisol	28%
	Urinary electrolytes	8%

A cortisol curve can be used to assess how appropriate dosing of glucocorticoid steroids in Addison's disease patients is

Important for me Less important

A cortisol day curve allows serial measurements to be taken to monitor how well cortisol is being replaced in patient with Addison's disease as well other causes of adrenal insufficiency such as panhypopituitarism. Morning serum cortisol is an appropriate investigation as first line testing for Addisons's disease, with Synacthen testing to confirm that diagnosis. Dexamethasone suppression testing can confirm Cushing's syndrome. Urinary electrolytes would be appropriate in the diagnosis of SIADH.

👍 👎 Discuss (5) Improve

Next question

Addison's disease: management ★

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:
- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:
- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards
- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 16

Addison's disease

Royal College of Physicians 👍 16 👎 8

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

ADDISON'S
DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 1 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **42.9%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓
14	✗

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Question 15 of 217

A 51-year-old lady librarian attends outpatient clinic with painful eyes. She reports that her vision has deteriorated over the past four weeks. On examination, she has proptosis, periorbital oedema and a painful complex ophthalmoplegia. She appears anxious and is worried about not coping at work. At present she smokes ten cigarettes daily.

What would be the most appropriate next step in managing this patient?

- ○ IV methylprednisolone ×
- ○ Surgical decompression ×
- ○ Smoking cessation advice ×
- ○ Total thyroidectomy ×
- ○ Artificial tear drops ×

Submit answer

Reference ranges

Score: **42.9%**

1	✗
2	✗
3	✗
4	✓
5	✗

6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓
14	✗
15	-

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Question 15 of 217 ✓

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What would be the most appropriate next step in managing this patient?

IV methylprednisolone	47%
Surgical decompression	6%
Smoking cessation advice	41%
Total thyroidectomy	3%
Artificial tear drops	2%

IV methylprednisolone is the treatment of choice for moderately severe active Graves' ophthalmopathy. IV steroids have fewer side effects than oral steroids. If symptoms or vision do not improve then urgent surgical decompression should be considered.

Artificial tear drops are useful for symptomatic relief.

Total thyroidectomy has shown no benefit in the treatment of thyroid eye disease.

Outcomes have been shown to be worse in those patients who smoke, therefore smoking cessation advice should be given.

Discuss (5) | Improve

Thyroid eye disease ★

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology
- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention
- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features
- the patient may be eu-, hypo- or hyperthyroid at the time of presentation
- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management
- smoking cessation
- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids

- radiotherapy
- surgery

Complications

Exposure keratopathy
- this is the most common complication of thyroid eye disease
- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy
- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia
- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)
- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):
- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes

- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

AI + Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy 👍 9 👎 8

Management of Graves' Orbitopathy

Suggest link

Report broken link

Score: **46.7%**

1 ✗

2 ✗

3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓
14	✗
15	✓

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A 52-year-old lorry driver was referred to the secondary care diabetes mellitus by his GP with poorly controlled diabetes. He was diagnosed with type 2 diabetes mellitus six years ago. Unfortunately, he has not engaged with lifestyle interventions and his diabetes has been poorly controlled since diagnosis. He has since developed diabetic nephropathy and proliferative retinopathy. Other than diabetes he has a past medical history comprising of ischaemic heart disease, hypertension, hypercholesterolaemia, osteoarthritis and gout. At the point of referral he was prescribed aspirin 75mg OD, ramipril 10mg OD, simvastatin 40mg ON, naproxen 250mg BD, co-codamol 8/500 2tabs QDS, lansoprazole 30mg OD, metformin 500mg TDS, gliclazide 80mg BD and pioglitazone 30mg OD. He smoked 20 cigarettes per day and consumed 15 units of alcohol per week.

Examination revealed an obese gentleman with a BMI of 38 kg/m. His blood pressure was 148/88 mmHg. Cardiovascular examination revealed the presence of normal heart sounds and a JVP of 3cm. Examination of the respiratory, abdominal and neurological systems was unremarkable. Investigations reveal the following results:

Na^+	136 mmol/l
K^+	5.1 mmol/l
Urea	14.1 mmol/l
Creatinine	148 μmol/l
Total cholesterol	5.1 mmol/l
HDL cholesterol	1.3 mmol/l
HbA1c	68 mmol/mol (8.4%)

Dr. Jamil Alrajhi

What is the next best step management step?

○ Commence exenatide ✕

○ Commence sitagliptin ×

○ Commence insulin glargine ×

○ Increase dose of pioglitazone ×

○ Commence orlistat ×

Submit answer

Reference ranges ﹀

Score: **46.7%**

1	✗
2	✗
3	✗
4	✓
5	✗
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13	✓
14	✗
15	✓
16	-

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Total cholesterol	5.1 mmol/l
HDL cholesterol	1.3 mmol/l
HbA1c	68 mmol/mol (8.4%)

What is the next best step management step?

Commence exenatide 71%

Commence sitagliptin	11%
Commence insulin glargine	14%
Increase dose of pioglitazone	1%
Commence orlistat	3%

This gentleman fulfills the NICE guidelines criteria for exenatide therapy. He is obese with poorly controlled diabetes despite multiple oral hypoglycaemic agents and is suffering from other morbidities relating to his weight. Exenatide may help facilitate weight loss and does not cause hypoglycaemia. Insulin is the other option here, but it may lead to weight gain. Furthermore, owing to his occupation as a lorry driver he would require a period of close observation of his blood sugar levels prior to being allowed to resume HGV driving. Exenatide is therefore the best option.

Discuss (13) Improve

Next question

Diabetes mellitus: management of type 2

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)

Management of T2DM	HbA1c target
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

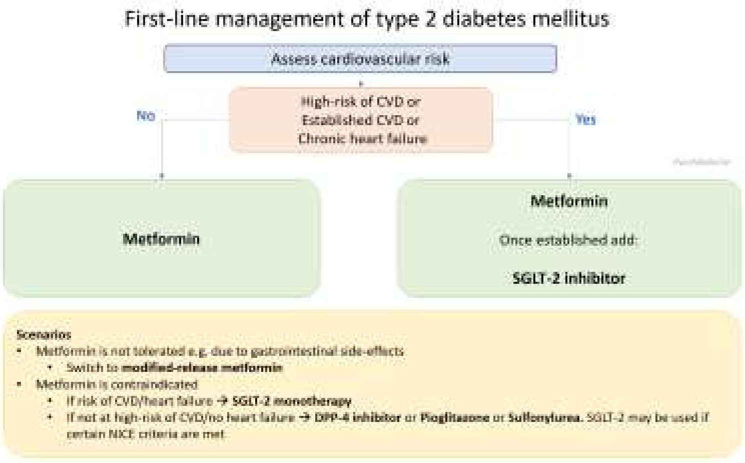
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios

- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

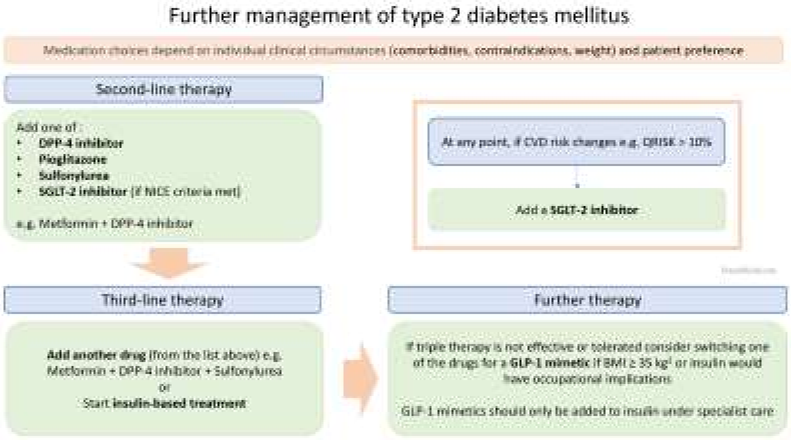
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

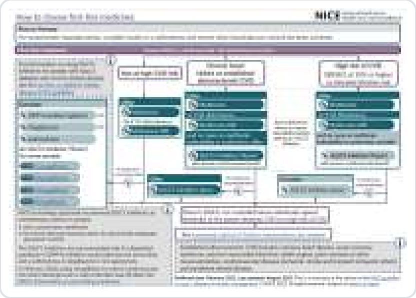
Starting insulin

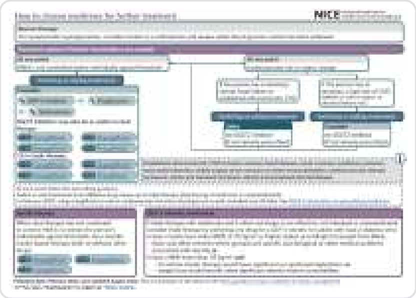
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea

Type 2 diabetes in adults: choosing medicines





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

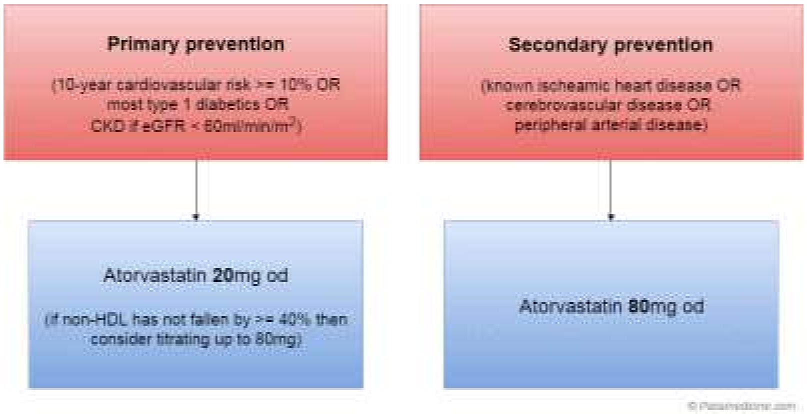
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

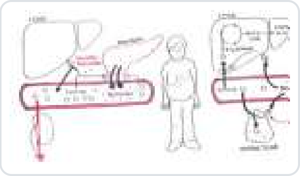
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

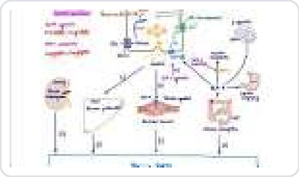
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **43.8%**

1	✗
2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
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12	✗
13	✓
14	✗
15	✓

16 ✗

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Question 17 of 217

A 42-year-old man presents for review in the endocrinology clinic. He has returned following investigations for hyperthyroidism as requested by the consultant. He originally presented to the emergency department with palpitations and was diagnosed with atrial fibrillations, and further investigations revealed an undetectable TSH with a free T4 of 52ng/dl. He was started on bisoprolol and apixaban since his discharge and has found his palpitations have improved significantly, but he still has them. On examination, he has no obvious neck swelling or tenderness. He returns after having had thyroid-stimulating hormone receptor antibodies tested. These are reported as undetectable. What is the next appropriate investigation?

- ○ Trial of carbimazole ×
- ○ Scan of radionuclide thyroid uptake ×
- ○ US-guided fine needle aspiration cytology ×
- ○ CT scan of the neck ×
- ○ Holter monitor for 48 hours ×

Submit answer

Dr. Jamil Alrajhi

Reference ranges

Score: **43.8%**

1 ✕

2	✗
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✓
11	✗
12	✗
13	✓
14	✗
15	✓
16	✗
17	-

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← Question 17 of 217 ✗ →

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🟥	Trial of carbimazole	22%
🟩	Scan of radionuclide thyroid uptake	73%
	US-guided fine needle aspiration cytology	3%
	CT scan of the neck	1%
	Holter monitor for 48 hours	1%

In hyperthyroidism, if TSH antibodies are negative then a radionuclide uptake scan can distinguish between types of hyperthyroidism

Important for me Less important

This patient has a typical presentation of hyperthyroidism, There is also biochemical evidence of hyperthyroidism, but autoantibodies for Grave's disease are negative. Thyroid-stimulating hormone receptor antibodies (TSH-RAb, commonly shortened to TRAb) has an up to 98% sensitivity and 99% specificity for Graves' disease. If this test is negative, then a radionuclide thyroid uptake scan can help determine the cause. In

Graves' disease there is diffuse high uptake, in thyroiditis there is low uptake, and if there are nodules then there is uneven uptake. US-guided fine needle aspiration cytology is recommended in the presence of features suggestive of malignancy. CT scan of the neck would only be appropriate in the case of a large goitre of which the limits would not be noted on examination or ultrasound.

Carbimazole treatment would only be appropriate as a short-term treatment awaiting radio-iodine treatment, in the medium term to induce remission in confirmed Graves' disease, and in the long-term where surgery and radio-iodine treatments are not an option. Whilst awaiting results, it would be more appropriate to up-titrate beta-blockers to gain symptomatic relief, rather than give carbimazole without a confirmed diagnosis.

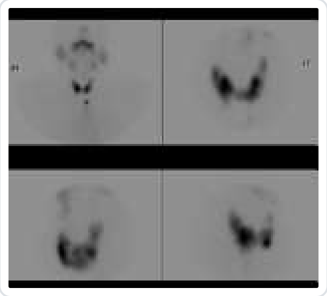
Discuss (4) Improve

Next question

Toxic multinodular goitre

Toxic multinodular goitre describes a thyroid gland that contains a number of autonomously functioning thyroid nodules resulting in hyperthyroidism.

Nuclear scintigraphy reveals patchy uptake.



The treatment of choice is radioiodine therapy.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **41.2%**

1	✗
2	✗
3	✗
4	✓
5	✗
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12	✗
13	✓
14	✗
15	✓

16 ✗

17 ✗

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Question 18 of 217

A 75-year-old man with a history of high blood pressure, type 2 diabetes and hypercholesterolaemia was admitted to the emergency department with confusion. His daughter states that this has come on slowly over the last week and prior to this he had no memory problems. He currently takes metformin, ramipril, amlodipine and atorvastatin.

On examination, he smells strongly of urine and his mucous membranes appear dry. His abbreviated mental test score is 7 out of 10 and he is oriented in person but not in place or time. His heart rate is 95 per minute and his blood pressure is 105/62 mmHg. His chest is clear and has a soft ejection systolic murmur which does not radiate. His jugular venous pressure is not visible and he has mild ankle oedema. He has diffuse tenderness in the lower abdomen with no peritonism and normal bowel sounds. He has no focal neurology.

Investigation results are as follows:

Chest x-ray: Clear lung fields.

Urine dip:

Glucose	+++
Blood	+
Protein	+
Leucocytes	+
Nitrites	+
Ketones	+

Venous blood gas:

pH	7.43
BE	- 1.5 mmol/l

HCO3	23 mmol/l
Glucose	34 mmol/l
Lactate	2.5 mmol/l

Full blood count:

Hb	120 g/l
Platelets	445 * 10^9/l
WBC	13 * 10^9/l

Renal function:

Na^+	151 mmol/l
K^+	5 mmol/l
Urea	10 mmol/l
Creatinine	137 μmol/l
Glucose	32 mmol/l
Ketones	2 mmol/l

Which would be the most appropriate initial resuscitation measure?

- ○ 0.45% saline ✕
- ○ 0.9% saline ✕
- ○ Fixed rate insulin and 0.9% saline ✕
- ○ Hartmann's ✕
- ○ Sliding scale insulin and 0.9% saline ✕

Submit answer

Reference ranges

Score: **41.2%**

1	✗
2	✗
3	✗
4	✓
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18	-

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K$^+$	5 mmol/l
Urea	10 mmol/l
Creatinine	137 μmol/l
Glucose	32 mmol/l
Ketones	2 mmol/l

Which would be the most appropriate initial resuscitation measure?

0.45% saline	6%
[✓ 0.9% saline]	74%
Fixed rate insulin and 0.9% saline	14%
Hartmann's	1%
Sliding scale insulin and 0.9% saline	5%

This gentleman has hyperosmolar hyperglycaemic state (HHS), likely precipitated by urinary tract infection and his pre-existing diabetes.

According to the Joint British Diabetes Society Guidelines for HHS, 0.9%

saline is the recommended initial resuscitation fluid, aiming for 3-6 litres positive at 12 hours. This should only be switched to 0.45% saline if osmolality is not declining despite positive fluid balance. Fixed rate insulin should only be added if glucose fails to fall with fluid.

Reference: Joint British Diabetes Societies Inpatient Care Group. The management of hyperosmolar hyperglycaemic state (HHS) in adults with diabetes. 2012.

👍 👎 Discuss (8) Improve

Next question

Hyperosmolar hyperglycaemic state ★

Hyperosmolar hyperglycaemic state (HHS) is a medical emergency that can be difficult to manage and has a significant associated mortality (up to 20%). Hyperglycaemia results in osmotic diuresis, severe dehydration, and electrolyte deficiencies. HHS typically presents in the elderly with type 2 diabetes mellitus (T2DM).

Pathophysiology

- hyperglycaemia → ↑ serum osmolality → osmotic diuresis → severe volume depletion

Precipitating factors

- intercurrent illness
- dementia
- sedative drugs

Clinical features

- whilst DKA presents within hours of onset, HHS comes on over many days, and consequently, the dehydration and metabolic disturbances may be more extreme
- consequences of volume loss
  - clinical signs of dehydration
  - polyuria

    - polydipsia
- systemic
    - lethargy
    - nausea and vomiting
- neurological
    - altered level of consciousness
    - focal neurological deficits
- haematological
    - hyperviscosity (may result in myocardial infarctions, stroke and peripheral arterial thrombosis)

There are no precise **diagnostic criteria** but the following are typically seen

- hypovolaemia
- marked hyperglycaemia (>30 mmol/L)
- significantly raised serum osmolarity (> 320 mosmol/kg)
    - can be calculated by: 2 * Na^+ + glucose + urea
- no significant hyperketonaemia (<3 mmol/L)
- no significant acidosis (bicarbonate > 15 mmol/l or pH > 7.3 - acidosis can occur due to lactic acidosis or renal impairment)

Management

- fluid replacement
    - fluid losses in HHS are estimated to be between 100 - 220 ml/kg
    - IV 0.9% sodium chloride solution
    - typically given at 0.5 - 1 L/hour depending on clinical assessment
    - potassium levels should be monitored and added to fluids depending on the level
- insulin
    - should not be given unless blood glucose stops falling while giving IV fluids
- venous thromboembolism prophylaxis
    - patients are at risk of thrombosis due to hyperviscosity

Complications

- vascular complications may occur due to hyperviscosity:

  - such as myocardial infarction
  - stroke

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Diabetes Societies 20 13

The management of the hyperosmolar hyperglycaemic state (HHS)

Suggest link

Report broken link

Score: **44.4%**

1 ✗
2 ✗
3 ✗
4 ✓

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6	✓
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18	✓

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A 22-year-old woman attends the clinic with a 12-month history of infertility and irregular periods. She also complains of excess hair growth over her face and chest. Observations are as follows: heart rate 82 beats per minute, blood pressure 115/75 mmHg, respiratory rate 16 breaths per minute, SpO2 98% (on air), temperature 37.2°C.

Blood results are as follows:

Hb	125 g/L	Male: (135-180) Female: (115 - 160)
Platelets	228 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na$^+$	132 mmol/L	(135 - 145)
K$^+$	5.1 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Glucose	5.2 mmol/L	(4-7)
Early morning cortisol	140 nmol/l	(>350)

What investigation is required?

- ○ ACTH stimulation testing ×
- ○ Dexamethasone suppression test ×
- ○ Karyotyping ×
- ○ Serum prolactin ×
- ○ Transvaginal ultrasound ×

Submit answer

Reference ranges ⌄

Score: **44.4%**

1	✗
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8	✓
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19	-

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Creatinine	74 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Glucose	5.2 mmol/L	(4-7)
Early morning cortisol	140 nmol/l	(>350)

What investigation is required?

🟩 ACTH stimulation testing	54%
🟥 Dexamethasone suppression test	8%
Karyotyping	5%
Serum prolactin	16%
Transvaginal ultrasound	16%

ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

Important for me Less important

ACTH stimulation testing is correct. The clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH). This form is milder, more common, and often diagnosed in later childhood or early adulthood. Usually, women with non-classic CAH present with signs of excess androgens (i.e., excess body hair, early puberty, irregular periods). Hypoaldosteronism (e.g. hyponatraemia, hyperkalaemia, metabolic acidosis) can also occur. The biochemical features and low early morning cortisol in this case are highly suggestive of hypoaldosteronism.

Dexamethasone suppression test is incorrect. Although Cushing's disease remains within the differential diagnosis, there are no other clinical (e.g. hypertension, proximal myopathy, weight gain, easy bruising) or biochemical features (e.g. hyperglycaemia, hypernatraemia, hypokalaemia) to support this diagnosis. Furthermore, the low early morning cortisol makes this an unlikely diagnosis.

Karyotyping is incorrect. Although Turner's syndrome can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Serum prolactin is incorrect. Although hyperprolactinaemia can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Transvaginal ultrasound is incorrect. Although ovarian tumours can result in androgen excess, this usually results in virilization which is not present in this case. Furthermore, hypoaldosteronism would not be expected to occur with ovarian pathology.

👍 👎 Discuss (5) Improve

Next question

Congenital adrenal hyperplasia

Congenital adrenal hyperplasia (CAH) refers to a group of autosomal recessive disorders that impair adrenal steroid biosynthesis. The deficiency in cortisol production leads to compensatory overproduction of adrenocorticotropic hormone (ACTH) by the anterior pituitary. Elevated ACTH levels increase the production of adrenal androgens, which can result in the virilization of female infants and affect genital development.

Cause

- 21-hydroxylase deficiency (90%)
    - impairs the conversion of 17-hydroxyprogesterone to 11-deoxycortisol, leading to cortisol deficiency and excess androgen production
- 11-beta hydroxylase deficihttps://www.passmedicine.com/database/dbnotes.php#ency (5%)
    - results in hypertension due to excess deoxycorticosterone
- 17-hydroxylase deficiency (very rare)
    - leads to mineralocorticoid excess with low androgen and estrogen levels

Clinical features

- the symptoms and signs of CAH vary depending on the specific enzyme deficiency and the severity of the disorder. Common clinical presentations include:
- virilization:
    - female infants may present with ambiguous genitalia due to excessive androgen exposure in utero.
    - male infants appear normal at birth, which can delay diagnosis.
- salt-wasting crisis:
    - this severe form occurs in about 75% of cases with 21-hydroxylase deficiency
    - it is characterized by dehydration, hypotension, and electrolyte imbalances, and can be life-threatening if not treated promptly.
- precocious puberty: Excess androgens can lead to early development of secondary sexual characteristics in both males and

females.
- infertility: Adults with untreated CAH may experience fertility issues due to hormonal imbalances.
- height and growth abnormalities: Children with CAH often experience accelerated growth rates initially but may have a shorter adult stature due to early epiphyseal closure.

Diagnosis
- many countries screen newborns for CAH, looking for elevated levels of serum concentration of 17-hydroxyprogesterone (17OHP). This is however not yet done in the UK
- ACTH stimulation testing is used to confirm the diagnosis
    - evaluates the adrenal gland's response to ACTH, with abnormal increases in 17OHP indicating CAH

Management
- primarily involves glucocorticoid replacement to reduce ACTH levels and minimize adrenal androgen production
- in cases of mineralocorticoid deficiency fludrocortisone is also prescribed

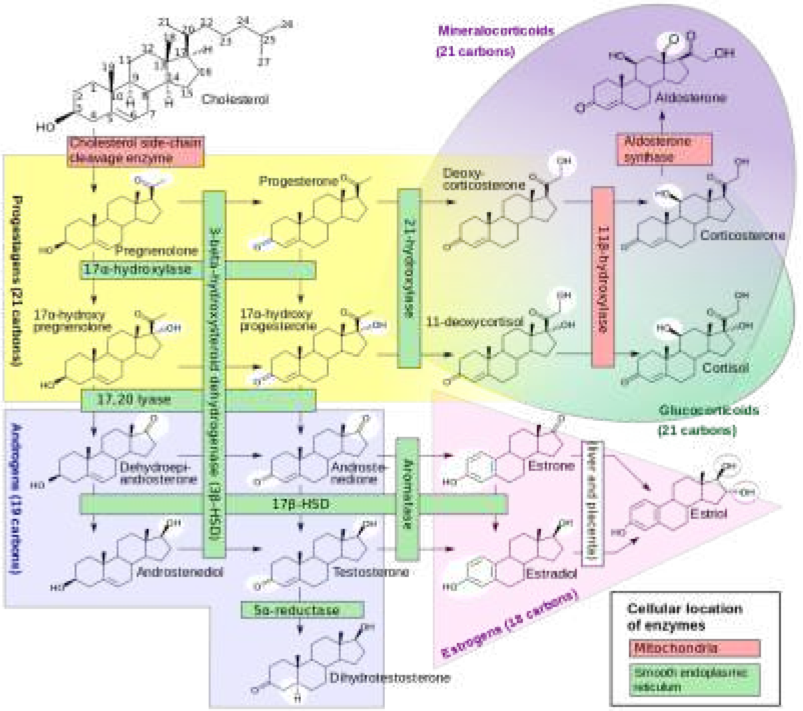

Cholesterol
HO
Cholesterol side-chain cleavage enzyme
Progestagens (21 carbons)
Pregnenolone
17α-hydroxylase
17α-hydroxy pregnenolone
OH
Progesterone
17α-hydroxy progesterone
3-beta-hydroxysteroid dehydrogenase (3β-HSD)
21-hydroxylase
17,20 lyase
Androgens (19 carbons)
Dehydroepi-androsterone
Androste-nedione
17β-HSD
Androstenediol
Testosterone
5α-reductase
Dihydrotestosterone
Mineralocorticoids (21 carbons)
Aldosterone
Aldosterone synthase
Deoxy-corticosterone
11β-hydroxylase
Corticosterone
11-deoxycortisol
Cortisol
Glucocorticoids (21 carbons)
Aromatase
Estrone
Estradiol
liver and placenta
Estriol
Estrogens (18 carbons)
Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

Q 123

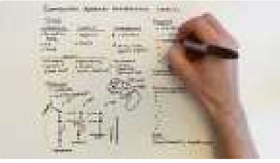
Next question

Textbooks

High-yield textbook

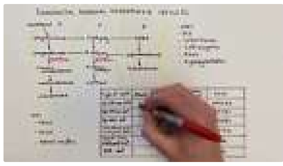
Extended textbook

Media



Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 18 2



Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 13 2

Report broken media

Score: **42.1%**

1	✗
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17	✗
18	✓
19	✗

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Question 20 of 217

A 35-year-old makes an urgent appointment in the diabetes clinic. She has had type 1 diabetes since the age of 20 but has controlled this well with insulin. She has no other medical problems and since diagnosed during a hospital admission with diabetic ketoacidosis has had no further hospital admissions or problems. She is concerned because she has fallen pregnant and wants to makes sure that she is not doing anything to harm the foetus. She is not looking to have a termination. She found out after she missed her period one week ago and took two pregnancy tests which were both positive. She had been taking a basal-bolus regime of insulin as well as atorvastatin. She had retinal screening and urine testing nine months ago. Her HbA1c was last measured three months ago at 39mmol/mol. She has been advised to continue with her insulin. She has also stopped the atorvastatin due to concerns about its effects in pregnancy. What is the most appropriate action to avoid complications of diabetes?

○ Restart atorvastatin ✕

○ Increase short-acting insulin dose ✕

○ Advise termination of pregnancy ✕

○ Refer for retinal screening ✕

○ Increase basal insulin dose ✕

Submit answer

Reference ranges

Score: **42.1%**

1	✗
2	✗
3	✗
4	✓
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20	-

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Question 20 of 217 ✓

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Restart atorvastatin	8%
Increase short-acting insulin dose	12%
Advise termination of pregnancy	1%
Refer for retinal screening	46%
Increase basal insulin dose	33%

Patients with diabetes should have increased frequency of retinal screening during pregnancy due to increased risk of retinopathy

Important for me Less important

The most appropriate plan is to repeat the retinal screening and test the urine. In diabetes, patients should achieve good diabetic control prior to planning for pregnancy. If this has not been achieved, then NICE advises contraception and to offer termination if pregnancy does occur due to increased risks in pregnancy. This patient has good diabetic control as evidenced by the lack of complications and HbA1c. When anticipating pregnancy, contraindicated drugs such as atorvastatin and ACE inhibitors should be stopped. Labetalol is an appropriate option for blood pressure control. The dose of insulin does not automatically need to be adjusted up or down but blood glucose would need close monitoring during pregnancy. It is advised, however, if the patient has not had retinal screening within the last six months to offer this urgently as there can be rapid development of diabetic retinopathy in pregnancy.

👍 👎 Discuss (7) Improve

Next question

Gestational diabetes

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:

- 87.5% have gestational diabetes
- 7.5% have type 1 diabetes
- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes

- BMI of > 30 kg/m^2
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes
- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)

- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:
- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose
- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
    - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
    - if glucose targets are still not met insulin should be added to diet/exercise/metformin
    - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios, insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets

with metformin but decline insulin treatment

Management of pre-existing diabetes
- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	Target
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link

Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

Report broken media

Score: **45%**

1 ✗
2 ✗
3 ✗
4 ✓

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19	✗
20	✓

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A 34-year-old woman presents for the first time as being 12 weeks pregnant. She has a past medical history of Hashimoto's thyroiditis. Her current medication is levothyroxine 100 mcg and recent blood tests reveal a TSH level of 1.0 mU/l.

What is the most appropriate management with regards to her levothyroxine treatment, given her recent diagnosis of pregnancy?

○ Reduce her levothyroxine dose by an average of 25 mcg ×

○ Increase her levothyroxine dose by an average of 100 mcg ×

○ Increase her levothyroxine dose by an average of 25-50 mcg ×

○ Reduce her levothyroxine dose by an average of 50 mcg ×

○ Keep her levothyroxine dose unchanged ×

Submit answer

Reference ranges

Score: **45%**

1 ✗
2 ✗
3 ✗
4 ✓

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21	-

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What is the most appropriate management with regards to her levothyroxine treatment, given her recent diagnosis of pregnancy?

Reduce her levothyroxine dose by an average of 25 mcg	2%
Increase her levothyroxine dose by an average of 100 mcg	2%
Increase her levothyroxine dose by an average of 25-50 mcg	88%
Reduce her levothyroxine dose by an average of 50 mcg	2%
Keep her levothyroxine dose unchanged	6%

In patients currently on levothyroxine who become pregnant, thyroid function tests should be assessed at 6-8 weeks gestation, 16-20 and at 28-32 weeks. During pregnancy, the average thyroxine requirements typically increase by 25-50 mcg. The patient normally returns to their original dose of levothyroxine straight after delivery.

Discuss (3) | Improve

Next question

Pregnancy: thyroid problems

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link Report broken link

Score: **47.6%**

1	✗
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15	✓
16	✗
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20	✓
21	✓

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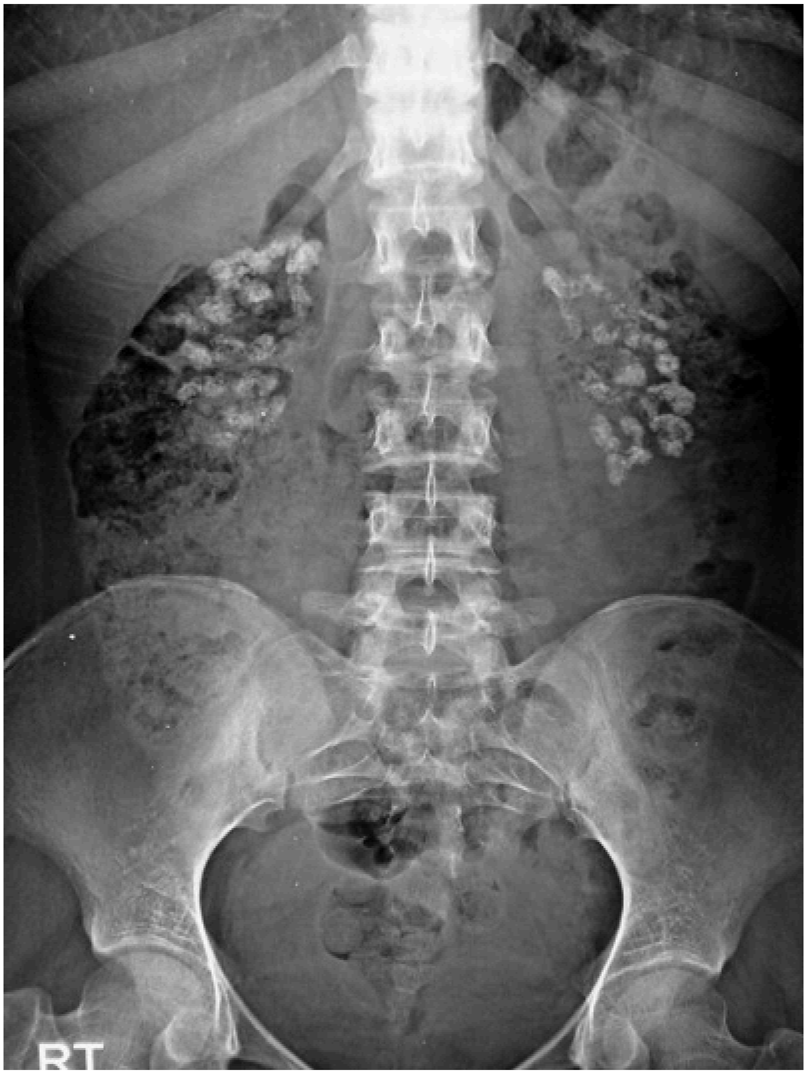
A 45-year-old woman presents to the Emergency Department with abdominal pain. Her GP is currently investigating her for lethargy, weakness and abdominal pain. Her symptoms have been getting progressively worse over the past few months. There is no past medical history of note. She smokes 5-10 cigarettes/day and drinks around 20 units of alcohol per week.

A urine dipstick has already been performed: protein trace, blood +, pH 5.5-6.0

Bloods show the following:

Hb	13.6 g/dl	Na^+	143 mmol/l
Platelets	$225 * 10^9/l$	K^+	2.3 mmol/l
WBC	$8.4 * 10^9/l$	Urea	6.1 mmol/l
Neuts	$6.0 * 10^9/l$	Creatinine	81 µmol/l
Lymphs	$1.9 * 10^9/l$	Bicarbonate	7 mmol/l
Eosin	$0.3 * 10^9/l$	Chloride	124 mmol/l

An abdominal film is requested due to her recurrent abdominal pains:



© Image used on license from Radiopaedia

What is the most likely diagnosis?

- ○ Renal tubular acidosis type 1 ×
- ○ Renal tubular acidosis type 2 ×
- ○ Renal tubular acidosis type 4 ×
- ○ Conn's syndrome ×
- ○ Bulimia ×

Submit answer

Reference ranges

Score: **47.6%**

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← Question 22 of 217 ✓ →

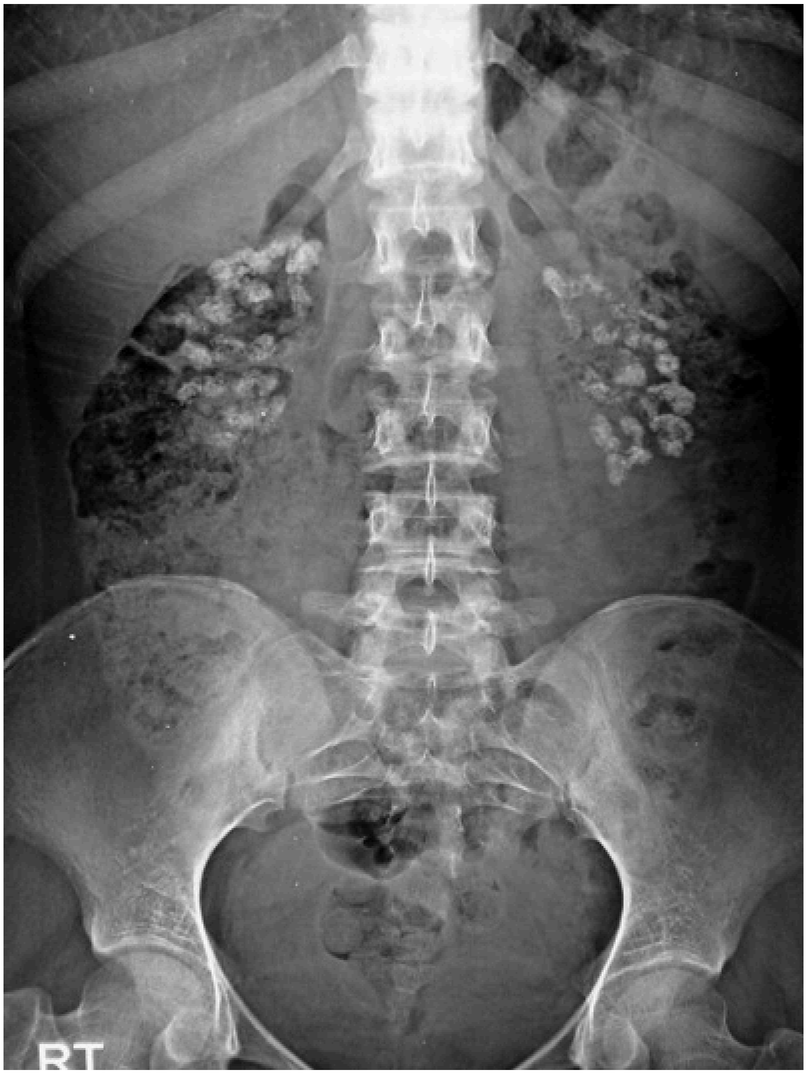
A 45-year-old woman presents to the Emergency Department with abdominal pain. Her GP is currently investigating her for lethargy, weakness and abdominal pain. Her symptoms have been getting progressively worse over the past few months. There is no past medical history of note. She smokes 5-10 cigarettes/day and drinks around 20 units of alcohol per week.

A urine dipstick has already been performed: protein trace, blood +, pH 5.5-6.0

Bloods show the following:

Hb	13.6 g/dl	Na^+	143 mmol/l
Platelets	225 * 10^9/l	K^+	2.3 mmol/l
WBC	8.4 * 10^9/l	Urea	6.1 mmol/l
Neuts	6.0 * 10^9/l	Creatinine	81 µmol/l
Lymphs	1.9 * 10^9/l	Bicarbonate	7 mmol/l
Eosin	0.3 * 10^9/l	Chloride	124 mmol/l

An abdominal film is requested due to her recurrent abdominal pains:



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What is the most likely diagnosis?

Renal tubular acidosis type 1	84%
Renal tubular acidosis type 2	11%
Renal tubular acidosis type 4	3%
Conn's syndrome	1%
Bulimia	0%

Hypokalaemia, nephrocalcinosis - type 1 renal tubular acidosis

Important for me Less important

This is probably a useful 'spot' diagnosis to learn for the exam: nephrocalcinosis on AXR → renal tubular acidosis (RTA) type 1. As well as nephrocalcinosis, the other pointers to RTA type 1 include hypokalaemia and a normal anion gap or hyperchloraemic metabolic acidosis.

If you weren't aware of the link between RTA type 1 and nephrocalcinosis another approach would be to work through the causes of 1. metabolic acidosis and 2. hypokalaemia.

1. Metabolic acidosis

It is useful to first calculate the anion gap:

Anion gap = (sodium + potassium) - (bicarbonate + chloride)

= (143 + 2.3) - (7 + 124) = 12.3 mmol/l

A normal anion gap is 8-14 mmol/l

Causes of a normal anion gap or hyperchloraemic metabolic acidosis	Causes of a raised anion gap metabolic acidosis
• gastrointestinal bicarbonate loss: diarrhoea, ureterosigmoidostomy, fistula • renal tubular acidosis • drugs: e.g. acetazolamide • ammonium chloride injection • Addison's disease	• lactate: shock, hypoxia • ketones: diabetic ketoacidosis, alcohol • urate: renal failure • acid poisoning: salicylates, methanol

2. Hypokalaemia

Potassium and hydrogen can be thought of as competitors. Hyperkalaemia tends to be associated with acidosis because as potassium levels rise fewer hydrogen ions can enter the cells

Hypokalaemia with alkalosis	Hypokalaemia with acidosis
- vomiting - diuretics - Cushing's syndrome - Conn's syndrome (primary hyperaldosteronism)	- diarrhoea - renal tubular acidosis (types 1 and 2) - acetazolamide - partially treated diabetic ketoacidosis

Discuss (2) Improve

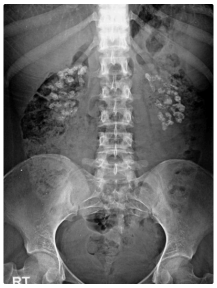
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule

- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

AI + Q 123

Next question

Textbooks

High-yield textbook

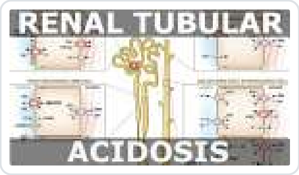
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **50%**

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A 48-year-old man attends the clinic for review. He complains of tiredness and weakness. He has a past medical history of alcoholism and currently drinks 88 units of beer per week. He takes regular thiamine. He is euvolaemic on examination.

Blood results are as follows:

Hb	122 g/l	Na^+	128 mmol/l
Platelets	385 * 10^9/l	K^+	3.6 mmol/l
WBC	8.5 * 10^9/l	Urea	3.2 mmol/l
Neuts	4.6 * 10^9/l	Creatinine	38 µmol/l
Lymphs	2.2 * 10^9/l	CRP	8 mg/l

A paired serum and urine test is as follows:

Serum osmolarity	271mOsm/kg (normal range 285-295)
Urine osmolarity	50 mOsm/kg (low)
Urinary sodium	8 mmol/l (low)

What is the most likely diagnosis?

○ Hypothyroidism ✕

○ Potomania ✕

○ SIADH ✕

○ Addison's disease ✕

○ Diabetes insipidus ✕

Submit answer

Reference ranges ⌄

Score: **50%**

1	✗
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23	-

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← Question 23 of 217 ✗ →

A 48-year-old man attends the clinic for review. He complains of tiredness and weakness. He has a past medical history of alcoholism and currently drinks 88 units of beer per week. He takes regular thiamine. He is euvolaemic on examination.

Blood results are as follows:

Hb	122 g/l	Na^+	128 mmol/l
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A paired serum and urine test is as follows:

Serum osmolarity	271mOsm/kg (normal range 285-295)
Urine osmolarity	50 mOsm/kg (low)
Urinary sodium	8 mmol/l (low)

What is the most likely diagnosis?

	Hypothyroidism	6%
🟩	Potomania	63%
🟥	SIADH	16%
	Addison's disease	3%
	Diabetes insipidus	12%

Beer potomania is a cause of hyponatremia which occurs due to a low dietary intake of solutes. Urine osmolarity will be low (<100 mosmol/kg) indicating that ADH is appropriately suppressed

Important for me Less important

Identifying the cause of hyponatraemia can be challenging. The first step is to confirm that it is a true hypotonic hyponatraemia. This is done by measuring the plasma osmolarity.

- If the measured plasma osmolarity is low this confirms true hypotonic hyponatraemia.
- If the plasma osmolarity is normal then this is suggestive of pseudohyponatraemia and should prompt you to measure proteins and lipids, which if present in high levels, can cause a pseudohyponatraemia due to the measuring technique.
- If the serum osmolarity is high then this confirms hypertonic hyponatraemia and should prompt you to check for high levels of solutes in the plasma, hyperglycaemia being the most common.

To identify the cause of the hyponatraemia there are several crucial considerations

- Volume status of the patient: hypervolaemic, euvolaemic, or hypovolaemic
- Paired serum and urine osmolarities
- Urinary sodium
- Thyroid and adrenal function
- History of patient e.g. diarrhoea, burns, vomiting, and kidney dysfunction. This is extremely important as may give clues to the cause (e.g. we could expect hypovolaemia and hyponatraemia with prolonged diarrhoea due to excessive loss of fluid and sodium from the GI tract)
- Drug history: several drugs are implicated in hyponatraemia through several mechanisms including SIADH

In this case, the low measured serum osmolarity confirms a true hypotonic hyponatraemia. The decreased plasma sodium can occur due to too much fluid (e.g. decreased urinary output or increased oral intake) or too little sodium (e.g. decreased oral intake or increased output through kidneys, GI tract, or skin). It can also be due to a combination of disturbed water and sodium balance e.g. combined sodium and water loss with a greater amount of sodium loss.

In this case, the patient is euvolaemic which narrows down the differential. Euvolamic hyponatraemia can occur due to:

- Hypothyroidism
- Psychogenic polydipsia
- Beer potomania

It is now very important to consider the urine osmolarity and sodium.

Urinary sodium is arguably the most useful biochemical measurement. Some key points include:

- In hyponatremia we would expect a low urinary sodium as the body attempts to conserve sodium
- Therefore a high urinary sodium >20 in the presence of hyponatraemia suggests renal sodium loss (e.g. CKD, diuretics, and salt losing nephropathy). Other causes of high urinary sodium include endocrine disease (e.g. Addison's), primary polydipsia, SIADH and reset osmostat
- A low urinary sodium (<20) in the presence of hyponatraemia suggest that the kidneys are conserving sodium. This should prompt you to consider extra-renal sodium losses (e.g. via GI tract or skin).
- Urinary sodium must be interpreted with caution. For example, if a patient is on diuretics the urinary sodium may be high after taking them but may go very low after their effects wear off, particularly if the patient is dehydrated
- The RAAS system is the dominant factor in sodium balance so consider this carefully
- In secondary hyperaldosteronism (e.g. heart failure, and cirrhosis) we get increased sodium retention and therefore a low urinary sodium
- In hypoaldosteronism (e.g. Addison's, true hypervolaemic states (e.g. polydipsia, SIADH)) we get high urinary sodium

Some key points with regards to urine osmolarity:

- In the presence of hyponatremia the bodies natural response to raising sodium would be to increase free water output resulting in a high urinary output with low urine osmolarity

- Urine osmolarity helps to differentiate between conditions associated with impaired free water excretion (high urine osmolarity >100), as seen in the majority of cases, and a few conditions associated with low urine osmolarity <100
- Urine osmolarity < 100 suggests that the kidneys are not concentrating urine e.g. ADH is suppressed
- Urine osmolarity > 100 suggests that ADH is not suppressed and is concentrating urine and impairing free water excretion
- Urine osmolarity will be raised in the majority of cases of hyponatraemia either due to inappropriate ADH release (e.g. SIADH), appropriate ADH release (hypovolaemia) or pseudo 'appropriate' ADH release (perceived intravascular depletion but actual hypervolaemic states e.g. heart failure and cirrhosis)
- With primary polydipsia, malnutrition (e.g. tea and toast syndrome, beer potomania) and a reset osmostat, the urine osmolarity is maximally dilute and generally less than 100

In this example, the history of excessive beer consumption, euvolaemic fluid status, low urine osmolarity, and a low urinary sodium, makes beer drinkers potomania the most likely cause of the hyponatraemia.

Potomania, also known as beer drinker's potomania is a specific hypo-osmolality syndrome related to massive consumption of beer, which is poor in solutes and electrolytes. With little food or other sources of electrolytes, consumption of large amounts of beer or other dilute alcoholic drinks leads a low level of urinary solute excretion (approximately 200-300 mOSM). This limits the amount of free water excretion to approximately 4 litres. Any intake above 4 litres would lead to a dilution of the serum sodium concentration and thus hyponatraemia.

Discuss (6) | Improve

Next question

Hyponatraemia ★

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)

- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic

- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss

- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

AI + Q 123

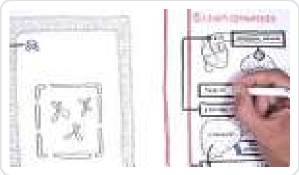
Next question

Textbooks

High-yield textbook

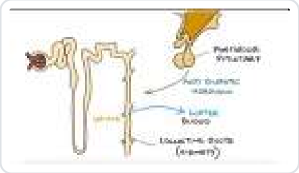
Extended textbook

Media



Hyponatraemia - classification, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 5 👎 0



Understanding Hyponatraemia

Zero To Finals - YouTube 👍 17 👎 1

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **47.8%**

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22	✓
23	✗

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A 72-year-old female presents with 5 days of general decline following a recent urinary tract infection, treated with oral antibiotics in the community by the GP. She is known to be a type 2 diabetic, diagnosed 28 years ago and insulin dependent for the past 6 years. She is normally on 46 units Lantus, 23 units TDS Novorapid. On examination, she is not orientated in time or place, GCS 14/15. She has no focal neurology, chest and cardiovascular auscultation are unremarkable. You demonstrate suprapubic tenderness on deep palpation but the abdomen is other soft and non-tender, bowel sounds are present. She appears extremely dehydrated: her mucous membranes are dry, peripheries cool with capillary refill time of 4 seconds and JVP +1 cm above the angle of Louis. Her blood sugar is 31mmol/L and a venous blood gas demonstrates pH 7.22, lactate 2 mmol/l, ketones 5 mmol/l. A urine dip is awaited. What is the most likely diagnosis?

- ○ Hyperglycaemic hyperketotic state ✕
- ○ Hyperglycaemia secondary to poor medical compliance during recent acute illness ✕
- ○ Urosepsis secondary to inadequately treated UTI ✕
- ○ Diabetic ketoacidosis ✕
- ○ Dehydration secondary to poor oral intake ✕

Submit answer

Reference ranges

Score: **47.8%**

1	✗
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Question 24 of 217 ✓

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Hyperglycaemic hyperketotic state	26%
Hyperglycaemia secondary to poor medical compliance during recent acute illness	5%
Urosepsis secondary to inadequately treated UTI	3%
Diabetic ketoacidosis	65%
Dehydration secondary to poor oral intake	0%

The patient is acidotic with ketones >3 mmol/l demonstrated, on a background of known insulin dependence. Although she is known to be a type 2 diabetic, it should be remembered that both types of diabetics can present as DKA, particularly advanced T2 DM who produce little to no endogenous insulin and are hence unable to shut down ketogenesis. Treatment should be as per DKA protocols, with intravenous fluids, fixed rate insulin infusion @ 0.1 unit/kg/hour, thromboprophylaxis, broad spectrum antibiotics and appropriate K+ replacement with insulin1.

Discuss (9) | Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management
- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids
  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and

- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

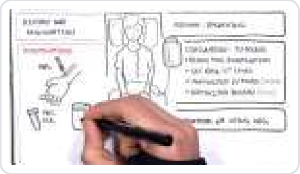
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

Report broken link

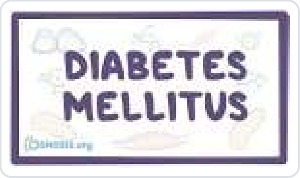
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **50%**

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Question 25 of 217

A 24-year-old woman is reviewed in the emergency department. She has presented with vomiting and feeling 'light-headed'. She is known to have a history of Addison's disease and has been unable to take her normal dose of hydrocortisone and fludrocortisone over the last five days due to nausea and vomiting. The past medical history includes hypothyroidism and vitiligo.

Her normal medication is:

- hydrocortisone 10mg, 10mg and 5mg at morning, lunchtime and afternoon
- fludrocortisone 100 micrograms
- levothyroxine 100 micrograms

So far she has been given hydrocortisone 100mg IV and 2L of IV fluids, as well as 10mg of metoclopramide IV. Her nausea has settled but she is still struggling to eat or drink. Her systolic blood pressure has increased from 82mmHg to 110mmHg. Capillary glucose is 8.5mmol/l.

She is prescribed regular IV hydrocortisone and anti-emetics.

What further prescription would be appropriate at this stage?

○ Start oral hydrocortisone ×

○ Immediate fludrocortisone ×

○ Start insulin on a sliding scale ×

○ Further IV fluids ×

○ Start IV antibiotics ×

Submit answer

Reference ranges

Score: **50%**

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- levothyroxine 100 micrograms

So far she has been given hydrocortisone 100mg IV and 2L of IV fluids, as well as 10mg of metoclopramide IV. Her nausea has settled but she is still struggling to eat or drink. Her systolic blood pressure has increased from 82mmHg to 110mmHg. Capillary glucose is 8.5mmol/l.

She is prescribed regular IV hydrocortisone and anti-emetics.

What further prescription would be appropriate at this stage?

	Start oral hydrocortisone	11%
🟥	Immediate fludrocortisone	36%
	Start insulin on a sliding scale	14%
🟩	Further IV fluids	34%
	Start IV antibiotics	5%

In an adrenal crisis, hydrocortisone is needed at high dose and fludrocortisone can be omitted as hydrocortisone has mineralocorticoid activity

Important for me Less important

This is a patient with known Addison's disease who presented unwell having been unable to take regular hydrocortisone doses. The key management is immediate IV hydrocortisone with plenty of IV fluids. When the patient is out of the immediate management stage, feeling better, and able to tolerate oral intake then hydrocortisone can be restarted orally. As this patient has not managed oral intake yet, prescribing oral hydrocortisone may lead to further missed doses. When the patient is having high dose IV hydrocortisone, there is significant mineralocorticoid activity, meaning that fludrocortisone is not needed. She is unlikely to need insulin at all, unless there is evidence of type 1 diabetes mellitus, which can be associated with Addison's disease.

👍 👎 Discuss (5) Improve

Next question

Addison's disease: management ★

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:

- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:

- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards

- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 19 16

Addison's disease

Royal College of Physicians 16 8

2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 1 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **48%**

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Question 26 of 217

You receive a phone call from a general practitioner regarding a 50 year-old man who has had thyroid function tests performed for a history of weight loss. There is no history of illicitly taking levothyroxine. His results show : TSH 0.01 mIU/L, T4 8.5 ug/dL. You should advise which of the following:

- ○ Admit for urgent MRI head ✕
- ○ Repeat the bloods and include parathyroid hormone (PTH) ✕
- ○ Add on T3 as this may represent T3 toxicosis ✕
- ○ Start radio-iodine treatment immediately ✕
- ○ Start thyroxine replacement ✕

Submit answer

Reference ranges

Score: **48%**

1	✕
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You receive a phone call from a general practitioner regarding a 50 year-old man who has had thyroid function tests performed for a history of weight loss. There is no history of illicitly taking levothyroxine. His results show : TSH 0.01 mIU/L, T4 8.5 ug/dL. You should advise which of the following:

Admit for urgent MRI head	12%
Repeat the bloods and include parathyroid hormone (PTH)	9%
Add on T3 as this may represent T3 toxicosis	71%
Start radio-iodine treatment immediately	3%
Start thyroxine replacement	5%

In approximately 5% of patients with clinical and biochemical hyperthyroidism T3 may be elevated prior to T4. This is known as T3 toxicosis.

When both free hormones are normal but TSH is low, the term subclinical thyrotoxicosis can be applied.

Radio-iodine treatment should never be started without discussion with the patient and is only used prior to carbimazole in certain circumstances. An MRI head would be part of the work up for secondary hyperthyroidism. PTH would not be useful in this circumstance and starting thyroxine would not be a good idea! T3 toxicosis is treated in the same manner as T4 hyperthyroidism.

Discuss (12) Improve

Next question

Subclinical hyperthyroidism

Subclinical hyperthyroidism is an entity which is gaining increasing recognition. It is defined as:

- normal serum free thyroxine and triiodothyronine levels
- with a thyroid stimulating hormone (TSH) below normal range (usually < 0.1 mu/l)

Causes

- multinodular goitre, particularly in elderly females
- excessive thyroxine may give a similar biochemical picture

The importance in recognising subclinical hyperthyroidism lies in the potential effect on the cardiovascular system (atrial fibrillation) and bone metabolism (osteoporosis). It may also impact on quality of life and increase the likelihood of dementia

Management

- TSH levels often revert to normal - therefore levels must be persistently low to warrant intervention
- a reasonable treatment option is a therapeutic trial of low-dose antithyroid agents for approximately 6 months in an effort to induce a remission

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **50%**

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Question 27 of 217

A 35-year-old patient presents to the emergency department after being brought in by ambulance. She had recently moved locally but had not yet registered with a GP, and therefore had an interruption in her supply of carbimazole for hyperthyroidism, diagnosed three months earlier. She is currently very unwell, complaining of palpitations. Clinically she has a raised JVP, bilateral crepitations on auscultation and severe peripheral oedema, and has become very breathless, and a temperature of 40.1 degrees C. She is started on oxygen. An ECG demonstrates a heart rate of 170bpm in AF, whilst a chest X-ray shows pulmonary oedema. What is the most appropriate immediate management?

○ IV propranolol ×

○ Propylthiouracil ×

○ Aspirin ×

○ Refer for emergency thyroid surgery ×

○ Potassium iodide ×

Submit answer

Reference ranges

Score: **50%**

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IV propranolol	80%
Propylthiouracil	16%
Aspirin	1%
Refer for emergency thyroid surgery	0%
Potassium iodide	3%

In thyroid storm with IV beta-blockers are a important first-line treatment

Important for me Less important

This is a patient with likely thyroid storm. The presentation suggests abrupt withdrawal of carbimazole has been the provoking factor, and now she has developed AF and heart failure. The most appropriate action is to rapidly control the heart rate by the use of IV beta-blockers. Propylthiouracil should also be started urgently, but the more critical action is to manage her heart rate. Aspirin may worsen the situation by displacing T4 from thyroid-binding globulin, causing an increase in free

T4. Whilst the patient will likely need both potassium iodide and thyroid surgery, neither are as urgent as controlling the heart rate.

👍 | 👎 | Discuss (7) | Improve

Next question

Thyroid storm ★

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:
- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **51.9%**

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A 16-year-old boy presents with several months of generalised weakness and muscle cramps. He also describes polydipsia and cravings for salty foods. He has no significant past medical history.

On examination, he appears generally well in himself. Observations are as follows:

- Respiratory rate of 18/min
- Blood pressure of 116/78mmHg
- Heart rate of 78/min

Blood tests are done and the results are as follows:

Na^+	129 mmol/L	(135 - 145)
K^+	2.8 mmol/L	(3.5 - 5.0)
Magnesium	0.54 mmol/L	(0.7 - 1.0)

A urine dipstick shows no glucose or protein present. A urine sample is sent to the lab for further analysis:

Urine calcium/creatinine ratio	0.05	(<0.14)

What is the most likely diagnosis?

○ Bartter syndrome ×

○ Dent disease ×

○ Fanconi syndrome ×

○ Gitelman syndrome ×

○ Liddle syndrome ×

Submit answer

Reference ranges ⌄

Score: **51.9%**

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K^+	2.8 mmol/L	(3.5 - 5.0)
Magnesium	0.54 mmol/L	(0.7 - 1.0)

A urine dipstick shows no glucose or protein present. A urine sample is sent to the lab for further analysis:

Urine calcium/creatinine ratio	0.05	(<0.14)

What is the most likely diagnosis?

Bartter syndrome	24%
Dent disease	2%
🟥 Fanconi syndrome	13%
🟩 Gitelman syndrome	57%
Liddle syndrome	5%

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

Important for me Less important

The syndrome presented here is that of **Gitelman syndrome**. This is a rare genetic disorder caused by mutations in the thiazide-sensitive sodium-chloride symporter in the distal convoluted tubule. Symptomatic patients present with symptoms identical to those taking excessive thiazide diuretics - hypokalaemia, hypomagnesaemia, and hyponatraemia. Patients are normotensive. Importantly, patients also display hypocalciuria, which differentiates the condition from Bartter syndrome. Treatment involves salt replacement.

Bartter syndrome is a similar condition, except that it involves a mutation in the Na/K/2Cl cotransporter in the loop of Henle. As such, the condition mimics loop diuretic abuse. Whilst also presenting with hypokalaemia, hypomagnesaemia, and normotension, the condition crucially presents with hypercalciuria, rather than hypocalciuria as seen in Gitelman syndrome.

Dent disease is a rare X-linked recessive condition that affects the proximal tubules. It is characterised by tubular proteinuria, hypercalciuria, and glycosuria. It is one of the causes of Fanconi syndrome. The scenario above is more closely aligned with Gitelman syndrome.

Fanconi syndrome is a syndrome of proximal tubule dysfunction. It presents with polyuria and polydipsia, like in this scenario, but patients would also have proteinuria and glycosuria, unlike the patient here. Hypokalaemia would also be noted, as well as hypophosphataemia. It may be genetic, such as being caused by Dent disease or acquired.

Liddle syndrome is an autosomal dominant condition - a mutation in the epithelial sodium channels (ENaC) of the kidneys, lungs, and sweat glands. The condition causes hypertension, which does not respond to medications, and hypokalaemia. Typically, children are asymptomatic at presentation. The patient in this scenario is normotensive.

Discuss (4) Improve

Next question

Gitelman's syndrome

Gitelman's syndrome is due to a defect in the thiazide-sensitive Na^+ Cl- transporter in the distal convoluted tubule.

Features

- normotension
- hypokalaemia
- hypocalciuria
- hypomagnesaemia
- metabolic alkalosis

Diagnosis

- genetic testing for mutations in the SLC12A3 gene

Management

- oral potassium and magnesium supplementation
- potassium-sparing diuretics may be used if still hypokalaemic/symptomatic

Q 123

Next question

Textbooks

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Extended textbook

Score: **50%**

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← Question 29 of 217 →

A 56-year-old man is referred to clinic by his General Practitioner as his GP had performed some routine blood tests which showed a K+ of 2.8 mmol/l. The patient feels well in himself. His past medical history includes angina and renal stones. On examination his chest is clear and his abdomen is soft and non-tender.

Observations are as follows: temperature 36.3, blood pressure 132/86 mmHg, heart rate 78/min, respiratory rate 16/min, saturations 95% on air

His ECG shows normal sinus rhythm.

Investigations are as follows:

Na+	142 mmol/l
K+	2.8 mmol/l
Creat	117 µmol/l
Urea	9.6 mmol/l
Urinary K+	26 mmol/l (normal <20)
PaO2	11.2 kPa
PaCO2	3.6 kPa
pH	7.32
HC03-	18 mmol/l
Base excess	-3 mmol/l

What is the most likely cause of his hypokalaemia?

- ○ Barter's Syndrome ✕
- ○ Liddle's Syndrome ✕
- ○ Renal tubular acidosis type 1 ✕

○ Renal tubular acidosis type 2 ✕

○ Renal tubular acidosis type 4 ✕

Submit answer

Reference ranges

Score: **50%**

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Question 29 of 217 ✓

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PaO2	11.2 kPa
PaCO2	3.6 kPa
pH	7.32
HC03-	18 mmol/l
Base excess	-3 mmol/l

What is the most likely cause of his hypokalaemia?

Barter's Syndrome	6%
Liddle's Syndrome	2%
Renal tubular acidosis type 1	79%

Renal tubular acidosis type 2	11%
Renal tubular acidosis type 4	2%

All of these diagnoses could cause hypokalaemia except renal tubular acidosis type 4 which causes a hyperkalaemia.

When faced with a patient with hypokalaemia first check the urinary K+, if this is low you should consider gastrointestinal losses such as diarrhoea or vomiting or decreased intake of K+. If the urinary K+ is high check the blood pressure, which is this case is normal. Liddle's Syndrome would cause hypertension.

If the patient is not hypertensive check the bicarbonate if it is low such as in this case the diagnosis is renal tubular acidosis. The clue here is that the patient has a history of renal stones meaning that the diagnosis is renal tubular acidosis type 1.

Renal tubular acidosis type 2 is associated with conditions such as Wilson's disease, lead poisoning and myeloma.

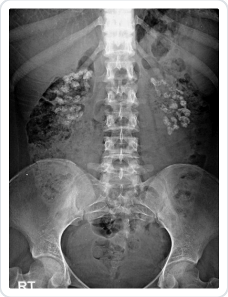
👍 👎 Discuss (5) Improve

Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

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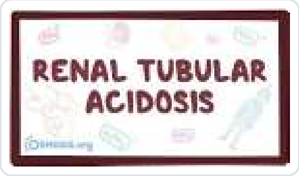
Extended textbook

Media


RENAL TUBULAR ACIDOSIS
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Score: **51.7%**

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Question 30 of 217

A 48-year-old female is admitted with an overdose of gliclazide. She states that she has taken ten 40mg tablets approximately three hours ago after getting into an argument with her boyfriend. She has a past medical history of type 2 diabetes mellitus, psychotic depression, and self-harm. Her drug history includes gliclazide and venlafaxine.

On examination, she appears sweaty and clammy and her heart rate is 120 bpm. Her Glasgow coma score is 13 (M6, V4, E3). Plasma glucose is 3.5 mmol/l.

You administer 100 ml of 20% dextrose. Her blood glucose initially rises to 5.5 mmol/l, however it rapidly drops back to 3.2 mmol/l shortly after, despite a continuing infusion of 20% dextrose.

What treatment will you give?

- ○ Lucozade ×
- ○ Complex carbohydrate ×
- ○ Reassurance ×
- ○ Insulin ×
- ○ Octreotide ×

Submit answer

Reference ranges

Score: **51.7%**

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Question 30 of 217 ✗

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You administer 100 ml of 20% dextrose. Her blood glucose initially rises to 5.5 mmol/l, however it rapidly drops back to 3.2 mmol/l shortly after, despite a continuing infusion of 20% dextrose.

What treatment will you give?

	Lucozade	4%
🟥	Complex carbohydrate	16%
	Reassurance	2%
	Insulin	1%
🟩	Octreotide	76%

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Important for me Less important

An overdose of sulphonylureas such as gliclazide can lead to severe and prolonged hypoglycaemia. The elimination half-life of gliclazide is long and varies between 12 and 20 hours. Gliclazide is readily absorbed from the gastrointestinal tract with peak plasma concentration occurring within 4 to 6 hours.

Octreotide is a somatostatin analogue which lowers insulin levels and thus raised blood glucose.

The patient had a reduced consciousness level, and it would, therefore, be inappropriate to administer Lucozade or complex carbohydrate.

Insulin would be entirely inappropriate as this would worsen the hypoglycaemia.

👍 👎 Discuss (3) Improve

Next question

Hypoglycaemia ★

Causes
- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
    - causes exaggerated insulin secretion
    - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia
- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later

- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic) neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features

- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
    - Sweating
    - Shaking
    - Hunger
    - Anxiety
    - Nausea
- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
    - Weakness
    - Vision changes
    - Confusion
    - Dizziness
- Severe and uncommon features of hypoglycaemia include:
    - Convulsion
    - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	C-peptide Level	Interpretation	Potential Causes
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
    - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form
    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
    - If the patient is alert, a quick-acting carbohydrate may be given (as above)
    - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
    - Alternatively, intravenous 20% glucose solution may be given through a large vein

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **50%**

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A 32-year-old alcoholic presents with abdominal pain and vomiting. His amylase is 1200 U/l and he is being treated for acute pancreatitis. You are called to see him as the nursing staff report the patient is becoming restless. He complains of numbness around his mouth and appears to be in some discomfort.

Your foundation year 2 colleague notes this morning's blood results:

Adjusted calcium	1.8mmol/l
Na^+	136 mmol/l
K^+	3.7 mmol/l
Urea	6.9 mmol/l
Creatinine	81 µmol/l

What's the next step in management?

○ Parathyroid hormone ×

○ Oral calcium supplementation ×

○ Calcitonin assay ×

○ Intravenous 10% calcium gluconate ×

○ Serum magnesium ×

Submit answer

Reference ranges

Score: **50%**

1	✗
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Question 31 of 217 ✓

A 32-year-old alcoholic presents with abdominal pain and vomiting. His amylase is 1200 U/l and he is being treated for acute pancreatitis. You are called to see him as the nursing staff report the patient is becoming restless. He complains of numbness around his mouth and appears to be in some discomfort.

Your foundation year 2 colleague notes this morning's blood results:

Adjusted calcium	1.8mmol/l
Na^+	136 mmol/l
K^+	3.7 mmol/l
Urea	6.9 mmol/l
Creatinine	81 µmol/l

What's the next step in management?

Parathyroid hormone	2%
Oral calcium supplementation	3%
Calcitonin assay	1%
[✓ Intravenous 10% calcium gluconate]	85%
Serum magnesium	9%

Intravenous calcium gluconate is used for the acute management of hypocalcaemia

Important for me Less important

In the acute setting of a patient with symptomatic hypocalcaemia intravenous replacement with 10% calcium gluconate would be the most sensible next step. Typically this is given over 10-30 minutes ideally with cardiac monitoring.

Checking the serum magnesium would be very useful as hypomagnesaemia would require replacement to also correct hypocalcaemia.

Oral calcium replacement would be an option in asymptomatic hypocalcaemic patients.

👍 | 👎 | 💬 Discuss (3) | Improve

Next question

Hypocalcaemia: causes and management ★

The clinical history combined with parathyroid hormone levels will reveal the cause of hypocalcaemia in the majority of cases

Causes
- vitamin D deficiency (osteomalacia)
- chronic kidney disease
- hypoparathyroidism (e.g. post thyroid/parathyroid surgery)
- pseudohypoparathyroidism (target cells insensitive to PTH)
- rhabdomyolysis (initial stages)
- magnesium deficiency (due to end organ PTH resistance)
- massive blood transfusion
- acute pancreatitis

Contamination of blood samples with EDTA may also give falsely low calcium levels.

Management
- severe hypocalcaemia (e.g. carpopedal spasm, tetany, seizures or prolonged QT interval) requires IV calcium replacement
    - the preferred method is with intravenous calcium gluconate, 10ml of 10% solution over 10 minutes

  - intravenous calcium chloride is more likely to cause local irritation
  - ECG monitoring is recommended
- further management depends on the further underlying cause

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 3 👎 2

Report broken media

Score: **51.6%**

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Question 32 of 217

A 59-year-old gentleman with capricious type 2 diabetes mellitus is reviewed in a community diabetic clinic. Despite optimising lifestyle and diet, glycaemic control is still poor on first line oral hypoglycaemic therapy. His diabetic consultant decides to start him on dapagliflozin 10mg daily.

Which of the following is a common side effect of dapagliflozin the patient should be warned about?

- ○ Pancytopaenia ×
- ○ Jaundice ×
- ○ Increased likelihood of pancreatitis ×
- ○ Increased likelihood of urinary infections ×
- ○ Increased risk of ischaemic heart disease ×

Submit answer

Reference ranges

Score: **51.6%**

1 ✗
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A 59-year-old gentleman with capricious type 2 diabetes mellitus is reviewed in a community diabetic clinic. Despite optimising lifestyle and diet, glycaemic control is still poor on first line oral hypoglycaemic therapy. His diabetic consultant decides to start him on dapagliflozin 10mg daily.

Which of the following is a common side effect of dapagliflozin the patient should be warned about?

Pancytopaenia	1%
Jaundice	1%
Increased likelihood of pancreatitis	7%
✓ Increased likelihood of urinary infections	91%
Increased risk of ischaemic heart disease	1%

Dapagliflozin is a SGLT-2 inhibitor which causes increased renal glucose loss to control diabetic glycaemia. It may cause recurrent urinary infections due to high urinary glucose load

Important for me Less important

Since the regulation of glucose and insulin is affected by many physiological variables, there are many targets for drugs to influence glycaemic homeostasis. Older oral hypoglycaemic drugs such as the sulphonylureas (gliclazide, etc) work by directly stimulating insulin release from pancreatic beta cells, whereas biguanides like metformin inhibit glucose mobilisation from hepatic stores and improve glucose handling. Metformin is associated with lactic acidosis and sulphonylureas are associated with increased incidence of hepatitis and jaundice.

A class of oral hypoglycaemic agent known as the thiazolidinediones (pioglitazone, etc) also improve glucose processing by increasing upregulation of genes which augment glucose and fat metabolism. This occurs by stimulation of the nuclear signalling protein peroxisome proliferator-activated receptor gamma (PPAR-). Thiazolidinediones have fallen out of vogue of late due to concerns about heart failure and ischaemic heart disease.

A more novel treatment for hyperglycaemia is the use of incretins which are gastrically derived peptides which also stimulate insulin secretion from beta cells. The two main peptides of use are glucose like peptide 1 (GLP-1) and gastric inhibitory peptide (GIP) both of which synergistically stimulate beta cell insulin release and inhibit alpha-cell glucagon release, causing a fall in blood glucose. Incretin drugs of this class include exenatide and liraglutide and are usually given as subcutaneous daily injections. In vivo, GLP-1 and GIP are rapidly deactivated by the enzyme dipeptidyl peptidase-4 (DPP-4) which limits their use (although the synthetic incretin analogue drugs are not deactivated by this enzyme). Drugs which can inhibit DPP-4 are useful in controlling blood glucose since they allow physiological gut incretins to stimulate insulin release. Examples of these drugs include sitagliptin, saxagliptin, etc. Rarely DPP-4 inhibitors and PPAR- modulators can cause hepatitis, pancreatitis and pancytopenia. Previous pancreatitis is a relative contraindication to their use.

Dapagliflozin is a renal SGLT-2 (sodium glucose transporter) inhibitor which has only recently obtained UK and US licensing authority. It causes an increase in glucose excretion by the kidney to lower serum glucose concentrations. SGLT-2 is a sodium/glucose cotransporter protein in the nephrons proximal tubule which reabsorbs glucose from the renal filtrate. These drugs cause heavy urinary glucose loss and can cause recurrent urinary infections and candidiasis as well as hypoglycaemia, crystalluria and renal failure due to osmotic diuresis.

👍 👎 Discuss (6) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail,*

for adults with type 2 diabetes'
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

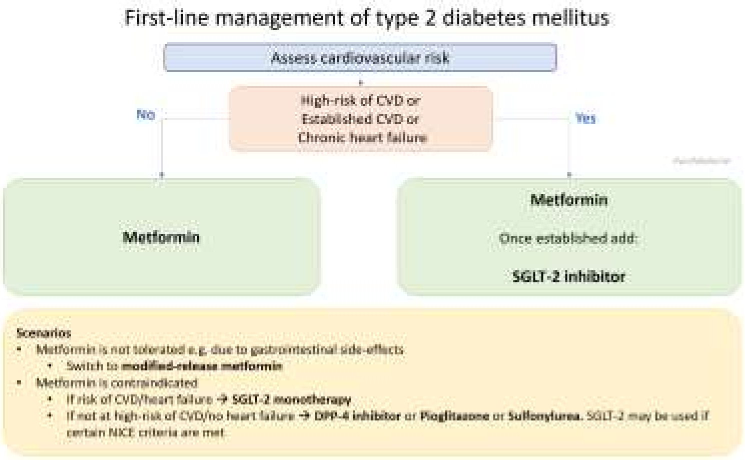
Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

Practical examples
- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

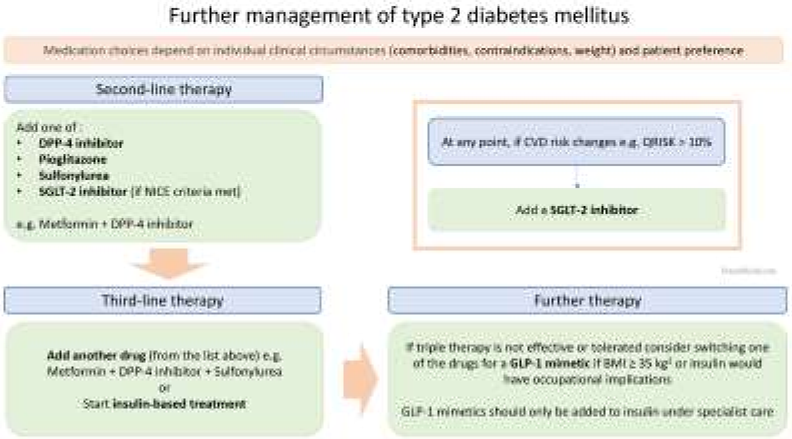
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:

    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea

- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

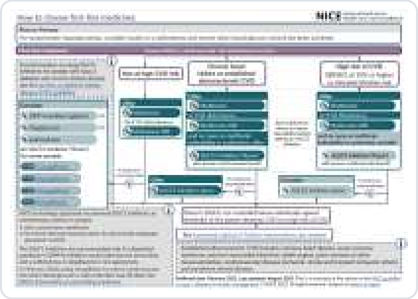
GLP-1 mimetics should only be added to insulin under specialist care

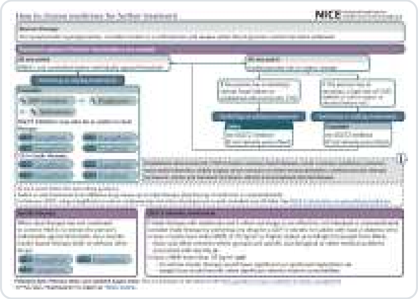
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

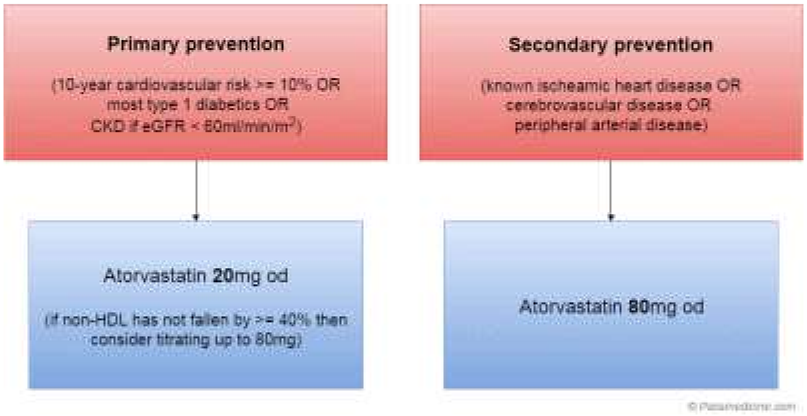
- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line

    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets
- should not be offered unless a patient has existing cardiovascular disease

Lipids
- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

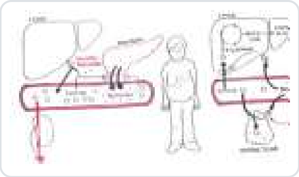
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

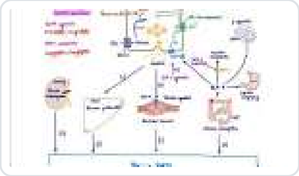
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **53.1%**

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← Question 33 of 217 →

A 25-year-old female presents with galactorrhoea. She reports no other symptoms on systematic enquiry. She has no past medical history and takes no regular medicines.

Blood results are as follows:

FSH	0.2 mIU/ml (normal 1 - 8)	LH	0.5 mIU/ml (normal 1-18)
Oestradiol	482 pg/ml (normal 27 - 123)	Progesterone	46 ng/mL (normal 5 - 20)
Prolactin	82 ng/dL (normal 5 - 40)		

What investigation will you perform?

- ○ MRI pituitary ×
- ○ Thyroid function tests ×
- ○ Pregnancy test ×
- ○ Pelvic ultrasound ×
- ○ No further investigations ×

Submit answer

Reference ranges ⌄

Score: **53.1%**

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Blood results are as follows:

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Oestradiol	482 pg/ml (normal 27 - 123)	Progesterone	46 ng/mL (normal 5 - 20)
Prolactin	82 ng/dL (normal 5 - 40)		

What investigation will you perform?

	MRI pituitary	21%
🟥	Thyroid function tests	7%
🟩	Pregnancy test	61%
	Pelvic ultrasound	10%
	No further investigations	1%

Pregnancy is an important differential to consider when assessing a female patient with hyperprolactinaemia

Important for me Less important

The hormone results are entirely consistent with pregnancy. Oestradiol and progesterone are secreted from the corpus luteum in early pregnancy. At approximately the third month of gestation, the placenta takes over as the source of oestrogen and progesterone. High levels of these hormones results in suppression of LH and FSH. During pregnancy, prolactin levels also increase. Prolactin causes enlargement of the mammary glands and prepares for milk production.

Discuss (6) Improve

Next question

Prolactin and galactorrhoea ★

Prolactin is secreted by the anterior pituitary gland with release being controlled by a wide variety of physiological factors. Dopamine acts as the primary prolactin releasing inhibitory factor and hence dopamine agonists such as bromocriptine may be used to control galactorrhoea. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia

Features of excess prolactin

- men: impotence, loss of libido, galactorrhoea
- women: amenorrhoea, galactorrhoea

Causes of raised prolactin

- prolactinoma
- pregnancy
- oestrogens
- physiological: stress, exercise, sleep
- acromegaly: 1/3 of patients
- polycystic ovarian syndrome
- primary hypothyroidism (due to thyrotrophin releasing hormone (TRH) stimulating prolactin release)

Drug causes of raised prolactin

- metoclopramide, domperidone
- phenothiazines
- haloperidol
- very rare: SSRIs, opioids

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 11 👎 5

Galactorrhoea review

Suggest link

Report broken link

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **51.5%**

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32 ✓

33 ✗

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Question 34 of 217

A 59-year-old man who has had type 1 diabetes mellitus for 40 years comes to the clinic for review. He has peripheral diabetic sensory neuropathy, impotence, and has received laser therapy for bilateral diabetic retinopathy. His main complaint that he is now suffering from unpredictable vomiting of undigested food. Despite modifying his diet he still suffers from the problem. On one occasion when he was admitted to hospital the condition responded well to erythromycin. Barium swallow has demonstrated significantly prolonged gastric emptying.

Which of the following is the most appropriate long-term intervention?

- ○ Ondansetron ×
- ○ Gastric pacemaker ×
- ○ Chlorpheniramine ×
- ○ Domperidone ×
- ○ Erythromycin ×

Submit answer

Reference ranges

Score: **51.5%**

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34	-

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A 59-year-old man who has had type 1 diabetes mellitus for 40 years comes to the clinic for review. He has peripheral diabetic sensory neuropathy, impotence, and has received laser therapy for bilateral diabetic retinopathy. His main complaint that he is now suffering from unpredictable vomiting of undigested food. Despite modifying his diet he still suffers from the problem. On one occasion when he was admitted to hospital the condition responded well to erythromycin. Barium swallow has demonstrated significantly prolonged gastric emptying.

Which of the following is the most appropriate long-term intervention?

	Ondansetron	4%
	Gastric pacemaker	24%
	Chlorpheniramine	2%
🟩	Domperidone	50%
🟥	Erythromycin	19%

Diabetic gastroparesis is best managed long term with domperidone

Important for me Less important

Pro-kinetic anti-emetics are the intervention of choice for the treatment of diabetic gastroparesis, the diagnosis here. Vomiting is often unpredictable and doesn't respond to dietary modification, making the condition extremely debilitating for sufferers. Domperidone, a dopamine D2 receptor antagonist is the main prospect for long-term intervention, as it doesn't have the neurological liabilities which are associated with metoclopramide use, it may be associated with galactorrhoea due to a rise in prolactin levels in some patients who take it.

Ondansetron, a centrally acting anti-emetic is largely ineffective for gastroparesis, erythromycin is used for acute episodes of diabetic gastroparesis, and gastric pacemaker therapy is designated a research therapy of last resort by NICE. Chlorpheniramine is an anti-histamine that has some anti-emetic properties, although it is predominantly a centrally acting agent.

👍 👎 💬 Discuss (7) Improve

Next question

Diabetic neuropathy ★

Peripheral neuropathy

Diabetes typically leads to sensory loss and not motor loss. Sensory loss typically results in a 'glove and stocking' distribution, with the lower legs affected first due to the length of the sensory neurons supplying this area. Painful diabetic neuropathy is also a common problem in clinical practice.

NICE updated it's guidance on the management of neuropathic pain in 2013. Diabetic neuropathy is now managed in the same way as other forms of neuropathic pain:

- first-line treatment: amitriptyline, duloxetine, gabapentin or pregabalin
- if the first-line drug treatment does not work try one of the other 3 drugs
- tramadol may be used as 'rescue therapy' for exacerbations of neuropathic pain
- topical capsaicin may be used for localised neuropathic pain (e.g. post-herpetic neuralgia)
- pain management clinics may be useful in patients with resistant problems

Gastrointestinal autonomic neuropathy

Gastroparesis

- occurs secondary to autonomic neuropathy
- symptoms include erratic blood glucose control, bloating and vomiting
- management options include metoclopramide, domperidone or erythromycin (prokinetic agents)

Chronic diarrhoea

- often occurs at night

Gastro-oesophageal reflux disease

- caused by decreased lower esophageal sphincter (LES) pressure

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 4

2013 Neuropathic pain guidelines

NICE 👍 13 👎 7

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Score: **50%**

1	✗
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34	✗

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Question 35 of 217

A 52 year-old woman presents with a two day history of nausea and fever. On admission she is confused and her husband states that she was recovering from a recent upper respiratory tract infection and sore throat. He also mentions she has previously been experiencing episodes of diarrhoea and palpitations over the last three months.

Examination reveals a temperature of 40.6ºC, pulse rate of 160 beats per minute and blood pressure of 110/70 mmHg. Her pulse is irregularly irregular. Heart sounds 1 and 2 are present with no added sounds, lung fields are clear and her abdomen is soft and none tender, with bowel sounds being present.

Blood tests are taken and reveal:

Hb	13.2 g/dL
Platelets	$180 * 10^9$/l
WBC	$10.2 * 10^9$/l
Na^+	135 mmol/l
K^+	4.2 mmol/l
Urea	7.2 mmol/l
Creatinine	132 µmol/l
Thyroid stimulating hormone (TSH)	0.03 mu/l
Free thyroxine (T4)	31 pmol/l
Total thyroxine (T4)	220 nmol/l

What is the most appropriate immediate treatment?

○ Propylthiouracil, corticosteroids and propranolol ×

○ Propylthiouracil and propranolol ×

○ Radio-iodine, corticosteroids and propranolol ×

○ Propylthiouracil and corticosteroids ×

○ Radio-iodine, propranolol and carbimazole ×

Submit answer

Reference ranges

Score: **50%**

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Question 35 of 217 ✗

A 52 year-old woman presents with a two day history of nausea and fever. On admission she is confused and her husband states that she was recovering from a recent upper respiratory tract infection and sore throat. He also mentions she has previously been experiencing episodes of diarrhoea and palpitations over the last three months.

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Creatinine	132 µmol/l
Thyroid stimulating hormone (TSH)	0.03 mu/l
Free thyroxine (T4)	31 pmol/l
Total thyroxine (T4)	220 nmol/l

What is the most appropriate immediate treatment?

🟩 Propylthiouracil, corticosteroids and propranolol	79%
🟥 Propylthiouracil and propranolol	16%

Radio-iodine, corticosteroids and propranolol 2%

Propylthiouracil and corticosteroids 2%

Radio-iodine, propranolol and carbimazole 2%

Thyrotoxic storm is treated with beta blockers, propylthiouracil and hydrocortisone

Important for me Less important

This patient is having a thyrotoxic storm (hyperthyroid crisis) a rare medical emergency that is caused by an exacerbation of hyperthyroidism and characterised by decompensation of one or more organ systems in people with untreated or poorly treated hyperthyroidism. The precipitating cause is most commonly infection, as with this case, although it is important to check for other causes. The patient above is in atrial fibrillation and shows signs of renal impairment due to dehydration. First line treatment for this medical emergency is propylthiouracil, corticosteroids and propranolol, although chlorpromazine can be added for severe anxiety.

Discuss (6) Improve

Next question

Thyroid storm

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:

- thyroid or non-thyroidal surgery
- trauma
- infection

- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5°C
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **48.6%**

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Question 36 of 217

A 69-year-old woman presents to the general practitioner. She reports that when she gets dressed she has noticed that she has been involuntarily leaking urine throughout the day however she denies any episodes of sudden urgency. Otherwise, she is fit and well and performs her activities of daily living independently.

What is the most appropriate first-line management?

- ○ Bladder retraining ✕
- ○ Mirabegron ✕
- ○ Oxybutynin ✕
- ○ Pelvic floor muscle training ✕
- ○ Tamsulosin ✕

Submit answer

Reference ranges

Score: **48.6%**

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Question 36 of 217 ✗

A 69-year-old woman presents to the general practitioner. She reports that when she gets dressed she has noticed that she has been involuntarily leaking urine throughout the day however she denies any episodes of sudden urgency. Otherwise, she is fit and well and performs her activities of daily living independently.

What is the most appropriate first-line management?

	Bladder retraining	22%
🟥	Mirabegron	5%
	Oxybutynin	5%
🟩	Pelvic floor muscle training	67%
	Tamsulosin	1%

Urinary incontinence - first-line treatment:

- urge incontinence: bladder retraining
- stress incontinence: pelvic floor muscle training

Important for me Less important

This woman is presenting with stress incontinence, a leaking of urine normally triggered by coughing, laughing, or sneezing. Although often associated with pelvic floor damage for example during childbirth, it can also present as a result of age-related changes. The first-line management for stress incontinence is **pelvic floor muscle training** which is the correct answer here.

Bladder retraining is useful in cases of urge incontinence however this patient denies any history of urinary urgency. Urge incontinence occurs due to an overactive bladder. Bladder retraining aims to regain control over the function of the bladder. As stress incontinence is related to weakened pelvic floor muscles, bladder retraining would not be as effective as pelvic floor muscle training in this case.

Mirabegron is a beta-3 agonist that relaxes the detrusor muscle of the bladder, increasing bladder capacity. As the primary problem with stress incontinence is related to the pelvic floor muscles, mirabegron would not be the first-line treatment in this case.

Oxybutynin is an antimuscarinic that is also prescribed for an overactive bladder. Again, this relaxes the detrusor muscles but as the primary pathology in stress incontinence is related to the pelvic floor muscles, this would not be the first-line management step here.

Tamsulosin is an alpha-1 antagonist which relaxes the smooth muscle in the bladder neck and urethra. This is useful in the treatment of overflow incontinence as a result of urethral obstruction for example from benign prostatic hyperplasia. It has no effect on the pelvic floor muscles which are weakened in stress incontinence and so has no role here.

👍 👎 Discuss (1) Improve

Next question

Urinary incontinence ★

Urinary incontinence (UI) is a common problem, affecting around 4-5% of the population. It is more common in elderly females.

Risk factors
- advancing age
- previous pregnancy and childbirth
- high body mass index
- hysterectomy
- family history

Classification

- overactive bladder (OAB)/urge incontinence
  - due to detrusor overactivity
  - the urge to urinate is quickly followed by uncontrollable leakage ranging from a few drops to complete bladder emptying
- stress incontinence: leaking small amounts when coughing or laughing
- mixed incontinence: both urge and stress
- overflow incontinence: due to bladder outlet obstruction, e.g. due to prostate enlargement
- functional incontinence
  - comorbid physical conditions impair the patient's ability to get to a bathroom in time
  - causes include dementia, sedating medication and injury/illness resulting in decreased ambulation

Initial investigation

- bladder diaries should be completed for a minimum of 3 days
- vaginal examination to exclude pelvic organ prolapse and ability to initiate voluntary contraction of pelvic floor muscles ('Kegel' exercises)
- urine dipstick and culture
- urodynamic studies
  - not routinely required before starting initial conservative treatment for urinary incontinence
  - may be considered if the diagnosis is uncertain, mixed incontinence is suspected or surgical intervention is planned

Management depends on whether urge or stress UI is the predominant picture

If urge incontinence is predominant:

- bladder retraining (lasts for a minimum of 6 weeks, the idea is to gradually increase the intervals between voiding)
- bladder stabilising drugs: antimuscarinics are first-line
  - NICE recommend oxybutynin (immediate release), tolterodine (immediate release) or darifenacin (once daily preparation)
  - avoid immediate-release oxybutynin in frail older women
  - solifenacin is an alternative antimuscarinic agent

- mirabegron (a beta-3 agonist) may be useful if there is concern about anticholinergic side-effects in frail elderly patients
- if troublesome nocturia is a feature, desmopressin may be considered
- for women with overactive bladder that has not responded to non-surgical management:
    - if urodynamic investigations determine that detrusor overactivity is causing her overactive bladder symptoms then consider botulinum toxin type A bladder wall injection

If stress incontinence is predominant:

- lifestyle measures, e.g. weight reduction, reducing caffeine intake
- pelvic floor muscle training
    - NICE recommend at least 8 contractions performed 3 times per day for a minimum of 3 months
- surgical procedures include:
    - mid-urethral tape procedures (still recommended by NICE but only after discussing risks and benefits)
    - intramural bulking agent injections
    - colposuspension
- duloxetine may be offered to women if they decline surgical procedures
    - a combined noradrenaline and serotonin reuptake inhibitor
    - mechanism of action: increased synaptic concentration of noradrenaline and serotonin within the pudendal nerve → increased stimulation of urethral striated muscles within the sphincter → enhanced contraction

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 23 👎 30

2019 urinary incontinence guidelines

Suggest link Report broken link

Score: **47.2%**

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← Question 37 of 217 →

A 28-year-old woman with well-controlled hypothyroidism on a stable dose of 110 micrograms of thyroxine daily presents to the clinic for a routine check-up. She reports good health overall and has no additional medical issues. She has recently found out she is pregnant, currently in the fourth week of gestation.

What is the most appropriate management advice for this patient?

- ○ Check TSH before making dose adjustments ×
- ○ Continue on the current dose ×
- ○ Increase dose to 165 micrograms per day ×
- ○ Increase dose to 180 micrograms per day ×
- ○ Increase dose to 200 micrograms per day ×

Submit answer

Reference ranges

Score: **47.2%**

1 ✗
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What is the most appropriate management advice for this patient?

🟥	Check TSH before making dose adjustments	7%
	Continue on the current dose	2%
🟩	Increase dose to 165 micrograms per day	84%
	Increase dose to 180 micrograms per day	4%
	Increase dose to 200 micrograms per day	3%

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

Important for me Less important

Increase dose to 165 micrograms per day is the correct answer.

During pregnancy, there is a physiological increase in thyroid hormone requirements to support maternal health and normal fetal development. Consequently, patients with hypothyroidism often require an adjustment of their levothyroxine dosage by up to 50% to satisfy the increased demands of both mother and fetus. This patient is currently on a daily dose of 110 mcg of levothyroxine. The recommended increment can be calculated as follows:

110 mcg x 50% = 55 mcg.

Therefore, the dose should be increased to a maximum of (110 + 55) = 165 mcg per day, which is the correct answer for this case.

Check TSH before making dose adjustments is incorrect. Rather than waiting for TSH test results, it is necessary to adjust thyroxine dosages during pregnancy to meet the increased requirements. Subsequent monitoring with TSH assays post-adjustment is recommended throughout the pregnancy to ensure a euthyroid state.

Continue on the current dose would not suffice as it fails to address the increased hormonal needs during pregnancy.

Increase dose to 180 micrograms per day is incorrect because this suggests a rise of 70 mcg from her baseline dosage, amounting to a disproportionate increase of approximately 63%, which overshoots standard recommendations.

Increase dose to 200 micrograms per day is also incorrect as it means an excessive enhancement of her baseline dosage by 90 mcg (an 81% increment), far exceeding the advised guidelines for dose escalation during pregnancy.

👍 👎 Discuss (2) Improve

Next question

Pregnancy: thyroid problems ★

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link Report broken link

Score: **45.9%**

1	✗
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A 24-year-old woman presents to hospital after collapsing while out shopping. On taking her observations, she has a temperature 37.2°C, a pulse rate of 78 per minute which is regular and normal in character and a blood pressure of 164/92 mmHg. Heart sounds 1 and 2 were present with no added sounds and his chest was clear on auscultation. Her abdomen was soft and non-tender with no organomegaly. Neurological examination was unremarkable. She has no past medical history of note and is on no regular medications.

Further blood tests reveal low renin and aldosterone levels, hypokalaemia and a serum bicarbonate of 30 mmol/l.

Which of the following is the most appropriate treatment for her condition?

- ○ Angiotensin converting enzyme inhibitor therapy ×
- ○ Bumetanide ×
- ○ Potassium replacement ×
- ○ Spironolactone ×
- ○ Amiloride ×

Submit answer

Reference ranges

Score: **45.9%**

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Back to top

Question 38 of 217 ✓

A 24-year-old woman presents to hospital after collapsing while out shopping. On taking her observations, she has a temperature 37.2°C, a pulse rate of 78 per minute which is regular and normal in character and a blood pressure of 164/92 mmHg. Heart sounds 1 and 2 were present with no added sounds and his chest was clear on auscultation. Her abdomen was soft and non-tender with no organomegaly. Neurological examination was unremarkable. She has no past medical history of note and is on no regular medications.

Further blood tests reveal low renin and aldosterone levels, hypokalaemia and a serum bicarbonate of 30 mmol/l.

Which of the following is the most appropriate treatment for her condition?

Angiotensin converting enzyme inhibitor therapy	4%
Bumetanide	3%
Potassium replacement	6%
Spironolactone	28%
Amiloride	59%

This woman has presented with the combination of hypokalaemic alkalosis, suppressed renin and aldosterone levels in the presence of hypertension indicates a diagnosis of Liddle syndrome. Hypertension and hypokalaemia respond well to amiloride. Spironolactone is not as effective as this medication acts on the mineralocorticoid receptor, as opposed to amiloride, which acts directly on the sodium channel.

Discuss (5) | Improve

Next question

Liddle's syndrome ★

Liddle's syndrome is a rare autosomal dominant condition that causes hypertension and hypokalaemic alkalosis. It is thought to be caused by disordered sodium channels in the distal tubules leading to increased reabsorption of sodium.

Treatment is with either amiloride or triamterene

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

LIDDLE'S
SYNDROME

Liddle's syndrome

The Medics Guide - YouTube 👍 16 👎 0

Report broken media

Score: **47.4%**

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A 65-year-old man is reviewed in clinic. He has a past medical history of an NSTEMI, hypercholesterolaemia, hypertension and depression. He is euvolaemic on examination. His drug history includes sertraline, bisoprolol, ramipril and furosemide.

Blood results are follows:

Hb	138 g/l	Na^+	126 mmol/l
Platelets	440 * 10^9/l	K^+	3.8 mmol/l
WBC	10.8 * 10^9/l	Urea	7.2 mmol/l
Glucose	6.5 mmol/l	Creatinine	86 µmol/l
Total cholesterol	6.5 * 10^9/l	Triglycerides (fasting)	12.5 mmol/L (normal < 1.7)

You perform a paired serum and urine osmolarity:

Serum osmolarity	290 mOsmol/kg (normal 275-295)
Urine osmolarity	600 mOsmol/kg
Urine sodium	40 mmol/l

What is the most likely cause of the hyponatraemia?

○ SIADH ×

○ Dilutional hyponatraemia secondary to heart failure ×

○ Pseudohyponatraemia ×

○ Furosemide ×

○ Hypothyroidism ×

Submit answer

Reference ranges

Score: **47.4%**

1	✗
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← Question 39 of 217 ✗ ⚐ →

A 65-year-old man is reviewed in clinic. He has a past medical history of an NSTEMI, hypercholesterolaemia, hypertension and depression. He is euvolaemic on examination. His drug history includes sertraline, bisoprolol, ramipril and furosemide.

Blood results are follows:

Hb	138 g/l	Na^+	126 mmol/l
Platelets	440 * 10^9/l	K^+	3.8 mmol/l
WBC	10.8 * 10^9/l	Urea	7.2 mmol/l
Glucose	6.5 mmol/l	Creatinine	86 µmol/l
Total cholesterol	6.5 * 10^9/l	Triglycerides (fasting)	12.5 mmol/L (normal < 1.7)

You perform a paired serum and urine osmolarity:

Serum osmolarity	290 mOsmol/kg (normal 275-295)
Urine osmolarity	600 mOsmol/kg
Urine sodium	40 mmol/l

What is the most likely cause of the hyponatraemia?

🟥	SIADH	33%
	Dilutional hyponatraemia secondary to heart failure	3%
🟩	Pseudohyponatraemia	53%
	Furosemide	9%
	Hypothyroidism	2%

Pseudohyponatraemia is characterised by a normal measured serum osmolarity, however the calculated osmolarity (based on an erroneously low plasma sodium result) is reduced. This results in a raised osmolar gap

Important for me Less important

Identifying the cause of hyponatraemia can be challenging. The first step is to confirm that it is a true hypotonic hyponatraemia. This is done by measuring the plasma osmolarity.

- If the measured plasma osmolarity is low this confirms true hypotonic hyponatraemia.
- If the plasma osmolarity is normal then this is suggestive of pseudohyponatraemia and should prompt you to measure proteins and lipids, which if present in high levels, can cause a pseudohyponatraemia due to the measuring technique.
- If the serum osmolarity is high then this confirms hypertonic hyponatraemia and should prompt you to check for high levels of solutes in the plasma, hyperglycaemia being the most common.

In this case, the normal measured serum osmolarity suggests pseudohyponatraemia. Pseudohyponatraemia occurs due to a measuring technique defect. Using standard techniques, serum sodium is measured as a ratio of sodium to plasma volume. If the patients plasma has high amounts of proteins or lipids, the plasma volume will be increased resulting in a measured hyponatraemia. This is not a true hyponatraemia as the actual ratio of sodium to plasma fluid will be normal. Common causes include hyperproteinaemia (e.g. TPN, IVIG) and hyperlipidaemia (in particular hypertriglyceridemia).

In this case, the measured serum osmolarity can be compared with the calculated serum osmolarity. The calculated serum osmolarity = 2Na + 2K + glucose + urea = 273.3 mOsmol/kg. The osmolar gap = measured osmolarity - calculated osmolarity = 290 - 273.3 = 16.7 (normal <10). A raised osmolar gap is suggestive of the presence of other osmotically active particles. In this case it is due to the presence of a significantly raised triglyceride level.

Discuss (6) Improve

Next question

Hyponatraemia

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)

- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic

- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss

- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

+ Q 123

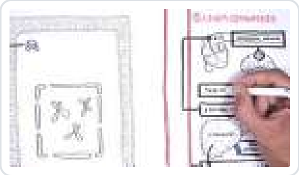
Next question

Textbooks

High-yield textbook

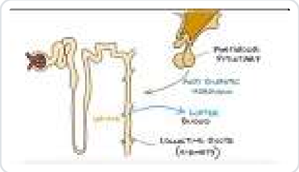
Extended textbook

Media



[Hyponatraemia - classification, causes, pathophysiology, treatment]

Armando Hasudungan - YouTube 👍 5 👎 0



[Understanding Hyponatraemia]

Zero To Finals - YouTube 👍 17 👎 1

WHAT IS...
SIADH?

[Syndrome of inappropriate antidiuretic hormone (SIADH)]

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **46.2%**

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Question 40 of 217

A 30-year-old woman with a history of type 1 diabetes presents to the Emergency department with nausea and vomiting coupled with increased urinary frequency over the past 3 days. She has been progressively losing weight and reducing her insulin dose after starting empagliflozin prescribed to help her lose weight and reduce glucose fluctuations. She also admits to taking a Chinese herbal remedy for weight control. Blood pressure is 100/70 mmHg, pulse is 88 beats per minute. pH is 7.25, glucose is 8.1 mmol/l, urine testing reveals ketones +++

Which of the following is the most likely diagnosis?

- ○ Empagliflozin related nephrotoxicity ×
- ○ Hyperosmolar non-ketotic state ×
- ○ Normoglycaemic ketoacidosis ×
- ○ Starvation ketoacidosis ×
- ○ Urinary sepsis ×

Submit answer

Reference ranges

Score: **46.2%**

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Which of the following is the most likely diagnosis?

Empagliflozin related nephrotoxicity	4%
Hyperosmolar non-ketotic state	1%
Normoglycaemic ketoacidosis	86%
Starvation ketoacidosis	9%
Urinary sepsis	1%

Hyperosmolar non-ketotic state is associated with marked hyperglycaemia and no ketosis. Starvation ketosis results in the formation of urinary ketones, but pH remains normal.

http://www.fda.gov/Drugs/DrugSafety/ucm446845.htm

Discuss (5) | Improve

Next question

Normoglycaemic ketoacidosis

Normoglycaemic ketoacidosis is increasingly recognised as a consequence of SGLT-2 inhibitor therapy in type 2 diabetes, and now more and more in patients with type 1 diabetes who are prescribed the drug off-license. The reason is thought to be that spilling of glucose into the urine leads to a reduction in plasma glucose and patients back off on insulin dose. They are then underdosed with respect to insulin, and SGLT-2 inhibitors also promote a rise in glucagon, which drives lipid oxidation and can further worsen the risk of ketoacidosis.

The problem can be avoided potentially by reducing the dose of the SGLT-2 inhibitor as patients reach blood glucose target.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **47.5%**

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Question 41 of 217

A 70-year-old man with a history of smoking 15 cigarettes/day presents with drowsiness, weight loss and a persistent cough. His investigations show:

Na^+	115 mmol/l	135-145 mmol/l
K^+	5.1 mmol/l	3.5 - 5.0 mmol/l
Urea	3 mmol/l	2.0-7 mmol/l
Creatinine	74 µmol/l	55-120 µmol/l

Plasma osmolality	270 mOsm/kg	285-295 mOsm/kg
Urine osmolality	1210	500 - 800 mOsm/kg

What is the most likely diagnosis?

- ○ Small cell lung cancer ×
- ○ Hypothyroidism ×
- ○ Encephalitis ×
- ○ Congestive cardiac failure ×
- ○ Squamous cell carcinoma ×

Submit answer

Reference ranges

Score: **47.5%**

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Urea	3 mmol/l	2.0-7 mmol/l
Creatinine	74 µmol/l	55-120 µmol/l

Plasma osmolality	270 mOsm/kg	285-295 mOsm/kg
Urine osmolality	1210	500 - 800 mOsm/kg

What is the most likely diagnosis?

Small cell lung cancer	92%
Hypothyroidism	0%
Encephalitis	1%
Congestive cardiac failure	1%
Squamous cell carcinoma	6%

A common endocrine complication of small cell lung cancer is SIADH

Important for me Less important

Hyponatraemia, reduced plasma osmolality and increased urine osmolality are suggestive of syndrome of inappropriate ADH secretion (SIADH).

The increase in ADH causes more aquaporin utilisation in the collecting duct system of the kidney. This causes more water to be retained, diluting the electrolytes in the blood and making the electrolytes in the urine more concentrated.

Small cell lung cancer is a common cause of SIADH and is the most likely diagnosis in this man with an extensive smoking history, cough and weight loss.

👍 | 👎 | Discuss (2) | Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology

- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water balance
- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels
- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.
- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension,

because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted, leading to hyponatraemia.

Causes of SIADH

Category	**Examples**
Malignancy	• **small cell lung cancer** • also: pancreas, prostate
Neurological	• stroke • subarachnoid haemorrhage • subdural haemorrhage • meningitis/encephalitis/abscess
Infections	• tuberculosis • pneumonia
Drugs	• sulfonylureas* • SSRIs, tricyclics • carbamazepine • vincristine • cyclophosphamide
Other causes	• positive end-expiratory pressure (PEEP) • porphyrias

Investigations
- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.
- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management

- correction must be done slowly to avoid precipitating central pontine myelinolysis
- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **48.8%**

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A 18-year-old man with type 1 diabetes and poor compliance attends hospital with shortness of breath, vomiting and feeling unwell. He is found to be in diabetic ketoacidosis and transferred to the high dependency unit. He has recovered well and is asymptomatic for a period but on day 3 of his treatment starts to feel unwell. He is tired and lethargic. His arms and legs ache and there is cramping at times. He walks to the bathroom and his legs collapse beneath him.

On examination, he is alert and has moist mucosa. There are no fasciculations or myoclonus. He has 4/5 power in all muscle groups with retained sensation. His abdomen is soft and his chest clear. A set of observations show that he is tachypnoeic at 24 breaths/min.

Bloods	admission	now
Na^+	128 mmol/l	133 mmol/l
K^+	6.1 mmol/l	4.5 mmol/l
Urea	9.2 mmol/l	5.6 mmol/l
Creatinine	134 μmol/l	87 μmol/l
Glucose	27.1mmol/l	12mmol/l
Ketones	3.1 mmol/l	0.2 mmol/l
pH	7.01	7.35

What is the likely course of his deterioration?

Anxiety	1%
🟩 Hypophosphataemia	55%
🟥 Cerebral pontine myelinolysis	22%
Sepsis	1%

Disequilibration syndrome 20%

Recovering DKA are at risk of hypophosphataemia

Important for me Less important

This gentleman has developed weakness following treatment for DKA. This is unlikely cerebral pontine myelinolysis as the sodium has been corrected by only 5mmol/l in 2-3 days. There are no focal signs suggesting sepsis though it is likely on the differential. Anxiety should only be made once other diagnoses are excluded. Disequilibration syndrome occurs in dialysis patients and is not relevant here. Therefore the answer is hypophosphataemia that often arises as a side effect of insulin with cells forming ATP and taking up free phosphate to achieve this.

👍 👎 Discuss (4) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed

insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management
- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low

    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	**Volume**
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	**Potassium replacement in mmol/L of infusion solution**
Over 5.5	Nil

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to

monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group 33 23

The Management of Diabetic ketoacidosis in adults

Suggest link

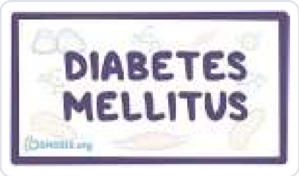
Report broken link

Media

Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2


DIABETES
MELLITUS

Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **47.6%**

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👍 👎 Discuss (4) Improve

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- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low

    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	**Volume**
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	**Potassium replacement in mmol/L of infusion solution**
Over 5.5	Nil

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to

monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

33 23

Suggest link

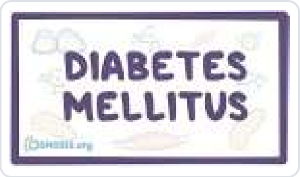
Report broken link

Media

Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2


DIABETES
MELLITUS

[Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms]

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **47.6%**

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43	-

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A 64-year-old man presents with confusion. A systematic enquiry was otherwise unremarkable. He has a history of bipolar disorder for which he takes lithium. He does not take any other regular medications. He drinks approximately 6 pints of beer per day. He is euvolemic on examination.

Blood results are as follows:

Na^+	125 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	72 µmol/L	(55 - 120)
Thyroid stimulating hormone (TSH)	4.8 mU/L	(0.5-5.5)
Free thyroxine (T4)	14.2 pmol/L	(9.0 - 18)
Early morning cortisol	440 nmol/l	(>350)

Osmolarity studies are requested:

Urine osmolarity	320 mmol/L	(300-900)
Urine sodium	54 mmol/L	(>40)
Plasma osmolarity	261.5 mOsm/kg	(275 - 295)

What is the most likely explanation?

○ Beer potomania ×

○ Cerebral salt wasting ×

○ Diabetes insipidus ×

○ Primary polydipsia ×

○ Syndrome of inappropriate antidiuretic hormone ADH release (SIADH) ×

Submit answer

Reference ranges ⌄

Score: **47.6%**

1	✗
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What is the most likely explanation?

Beer potomania	30%
Cerebral salt wasting	3%
Diabetes insipidus	12%
Primary polydipsia	7%

Syndrome of inappropriate antidiuretic hormone ADH release (SIADH) 48%

SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient

Important for me Less important

The patient has significant hyponatraemia. Since the serum osmolarity is < 275 we have confirmed a true hypotonic state.

In contrast, if the serum osmolarity was normal (275-295) then this would imply isotonic hyponatremia (i.e. pseudohyponatraemia). This is a measuring defect which can occur due to high levels of lipids (e.g. triglycerides) or proteins (e.g. macroglobulinaemia). These macromolecules take up a proportion of plasma volume thereby resulting in a spurious hyponatremia (as the lab measures Na / plasma volume rather than Na / plasma fluid).

If the serum osmolarity is > 295 then this implies a hypertonic hyponatremia. This occurs due to high levels of osmotically active solutes (e.g. hyperglycaemia or mannitol). This results in an osmotic shift of fluid into the extracellular space resulting in dilutional hyponatremia.

In this clinical case, we are dealing with a true hypotonic hyponatremia. The causes of this are wide but can broadly be divided into:

- Too much fluid (e.g. decreased output or increased intake)
- Too little sodium (e.g. decreased intake or increased output)
- Can also be due to a combination e.g. sodium and water loss (with sodium loss > water loss)

It is therefore absolutely essential to assess the fluid status of the patient to help identify the cause.

Urinary sodium is an exceptionally useful tool, and indeed after clinical examination, this is the most useful investigation. In hyponatremia, we would expect low urinary sodium as a physiological response to conserve sodium. Therefore urinary sodium >10 is suggestive of renal sodium loss (e.g. CKD, diuretics, hypoaldosteronism, and salt-losing nephropathy). Other causes of high urinary sodium include endocrine disease (e.g. Addison's), primary polydipsia, and SIADH. In SIADH, an initial hypervolaemic state results in aldosterone suppression resulting in natriuresis and high urinary sodium. In secondary hyperaldosteronism (e.g. heart failure, nephrotic syndrome) we get increased sodium retention and therefore low urinary sodium.

It is extremely important to note that urine osmolarities and electrolytes must be interpreted with caution. Urinary sodium will be high after taking diuretics, but may go very low after their effects wear off, particularly if the patient is dehydrated.

The urine osmolarity is often not particularly helpful. In the presence of hyponatremia, the body's natural response to raise sodium would be to increase free water output resulting in a high urinary output with low urine osmolarity. Therefore urine osmolarity helps to differentiate between conditions associated with impaired free water excretion (high urine osmolarity >100), as seen in the majority of cases, and a few conditions associated with low urine osmolarity <100.

The urine osmolarity will be raised (>100) in the majority of cases either due to inappropriate ADH release (e.g. SIADH), appropriate ADH release (hypovolaemia) or pseudo 'appropriate' ADH release (perceived intravascular depletion but actual hypervolaemic states e.g. nephrotic syndrome, heart failure and cirrhosis). However, if it is low (<100) then this suggests psychogenic polydipsia or beer drinker's protomania.

Syndrome of inappropriate antidiuretic hormone ADH release (SIADH) is correct. The patient fulfils the diagnostic criteria of SIADH making this the most likely diagnosis. Importantly he fulfils the biochemical criteria, has normal thyroid and adrenal function, is euvolemic, and is not on diuretics which may confound the biochemical analysis.

Beer potomania is incorrect. Potomania is a specific hypo-osmolality syndrome related to the massive consumption of beer, which is poor in solutes and electrolytes. In this condition, one would expect the urine osmolarity to be <100 since free water excretion is not impaired in this condition.

Cerebral salt wasting is incorrect. Both cerebral salt wasting and SIADH are characterised by hyponatremia with elevated urinary sodium and concentrated urine. The key distinguishing factor is that in cerebral salt wasting the patient is hypovolemic whereas SIADH patients are euvolaemic.

Diabetes insipidus is incorrect. The fact that the patient is on lithium is a red herring. Lithium is a well-known cause of diabetes insipidus (DI), however, DI is essentially the opposite condition to SIADH. In DI we have a failure of ADH to act on the kidneys resulting in polyuria, dehydration, raised serum osmolarity and hypernatraemia,

Primary polydipsia is incorrect. In this condition, one would expect the urine osmolarity to be <100 since free water excretion is not impaired.

👍 👎 Discuss (5) Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology

- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water balance
- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels

- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.
- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension, because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted, leading to hyponatraemia.

Causes of SIADH

Category	**Examples**
Malignancy	• **small cell lung cancer** • also: pancreas, prostate
Neurological	• stroke • subarachnoid haemorrhage • subdural haemorrhage • meningitis/encephalitis/abscess
Infections	• tuberculosis • pneumonia
Drugs	• sulfonylureas* • SSRIs, tricyclics • carbamazepine • vincristine • cyclophosphamide
Other causes	• positive end-expiratory pressure (PEEP) • porphyrias

Investigations

- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.
- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management
- correction must be done slowly to avoid precipitating central pontine myelinolysis
- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

+ Q 123

Next question

Textbooks

High-yield textbook

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Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **46.5%**

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Question 44 of 217

A 42-year-old woman is referred to the endocrine clinic. She is treated with lithium for bipolar disorder and presents with weight gain, lethargy, a dry cough and a hoarse voice over the past 3 months. Examination reveals patchy hair loss, a smooth goitre, and she is overweight with a body mass index of 32 kg/m². Her blood pressure is 122/82 mmHg, pulse is 60 beats per minute.

Investigations:

Na+	130 mmol/l
TSH	14.2 mIU/l

Which of the following is the most appropriate way to manage her?

○ Iodine supplementation ✕

○ Start prednisolone ✕

○ Stop lithium ✕

○ Surgical thyroidectomy ✕

○ Start thyroxine ✕

Submit answer

Reference ranges

Score: **46.5%**

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Question 44 of 217 ✗

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Investigations:

Na+	130 mmol/l
TSH	14.2 mIU/l

Which of the following is the most appropriate way to manage her?

	Iodine supplementation	5%
	Start prednisolone	7%
🟥	Stop lithium	37%
	Surgical thyroidectomy	3%
🟩	Start thyroxine	48%

Lithium increases intrathyroidal iodine content, inhibits the coupling of iodotyrosine residues to form iodothyronines (thyroxine and triiodothyronine), and it also inhibits release of T4 and T3 leading to hypothyroidism, the clinical picture seen here. The most appropriate intervention is thyroxine supplementation.

There is no need acutely to stop lithium therapy, although substitution may eventually be considered after discussion with the patient's psychiatrist. Due to the fact that lithium prevents coupling of iodotyrosine, iodine supplementation is ineffective. Prednisolone is also not of value because thyroiditis isn't the cause of the hypothyroidism seen here. Surgical thyroidectomy isn't indicated because there are no symptoms of extrinsic airway obstruction.

👍 👎 Discuss (8) Improve

Next question

Hypothyroidism: causes

Hypothyroidism affects around 1-2% of women in the UK and is around 5-10 times more common in females than males.

Primary hypothyroidism

Hashimoto's thyroiditis
- most common cause
- autoimmune disease, associated with IDDM, Addison's or pernicious anaemia
- may cause transient thyrotoxicosis in the acute phase
- 5-10 times more common in women

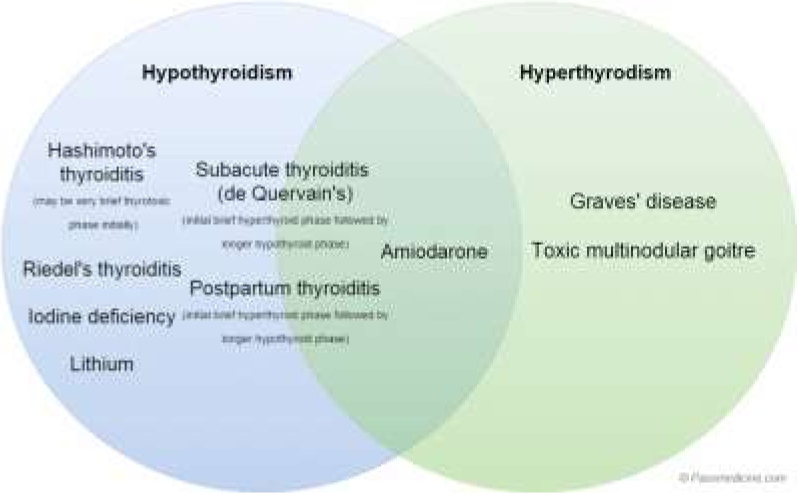
Subacute thyroiditis (de Quervain's)

Riedel thyroiditis

After thyroidectomy or radioiodine treatment

Drug therapy (e.g. lithium, amiodarone or anti-thyroid drugs such as carbimazole)

Dietary iodine deficiency


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Secondary hypothyroidism (rare)

From pituitary failure

Other associated conditions
- Down's syndrome
- Turner's syndrome
- coeliac disease

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Clinical features

Thyroid disease

Podmedics - YouTube 👍 1 👎 0

Hypothyroidism
Causes

Hypothyroidism and Hashimoto's Thyroiditis: Visual Explanation for Students

Zero To Finals - YouTube 👍 2 👎 1

Report broken media

Score: **45.5%**

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44 ✕

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Question 45 of 217

A 23-year-old woman attends a fertility clinic with her partner. She complains of oligomenorrhoea and galactorrhoea and has failed to get pregnant after 18 months of regular unprotected intercourse. Blood tests reveal a serum prolactin level of 6000 mIU/l (normal <500 mIU/l). A pituitary MRI is arranged which shows a microprolactinoma.

Which of the following is the best initial treatment?

- ○ Octreotide ×
- ○ Bromocriptine ×
- ○ Trans-sphenoidal hypophysectomy ×
- ○ Pituitary radiotherapy ×
- ○ Transfrontal hypophysectomy ×

Submit answer

Reference ranges

Score: **45.5%**

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Question 45 of 217

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- ○ Pituitary radiotherapy ×
- ○ Transfrontal hypophysectomy ×

Submit answer

Reference ranges

Score: **45.5%**

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Which of the following is the best initial treatment?

	Octreotide	10%
🟩	Bromocriptine	79%
🟥	Trans-sphenoidal hypophysectomy	10%
	Pituitary radiotherapy	1%
	Transfrontal hypophysectomy	1%

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

Important for me Less important

This patient has a prolactinoma. In the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. bromocriptine) which inhibit the release of prolactin from the pituitary gland. Surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension. Radiotherapy is rarely performed and octreotide is a somatostatin analogue used in the treatment of acromegaly.

👍 👎 Discuss (2) Improve

Next question

Prolactinoma

Prolactinomas are a type of pituitary adenoma, a benign tumour of the pituitary gland.

Pituitary adenomas can be classified according to:

- **size** (a microadenoma is <1cm and a macroadenoma is >1cm)
- **hormonal status** (a secretory/functioning adenoma produces and excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin.

Features

- excess prolactin in women
    - amenorrhoea
    - infertility
    - galactorrhoea
    - osteoporosis
- excess prolactin in men
    - impotence
    - loss of libido
    - galactorrhoea
- other symptoms may be seen with macroadenomas
    - headache.
    - visual disturbances (classically, a bitemporal hemianopia (lateral visual fields) or upper temporal quadrantanopia)
    - symptoms and signs of hypopituitarism

Diagnosis

- MRI

Management

- in the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. cabergoline, bromocriptine) which inhibit the release of prolactin from the pituitary gland
- surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 5 2

Report broken media

Score: **44.4%**

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Back to top

A 36-year-old woman is reviewed 3 months post-partum. During the pregnancy she was diagnosed with gestational diabetes. Following the delivery her glycaemic control has failed to improve and she has been diagnosed as having type 2 diabetes mellitus. She is only slightly overweight (body mass index 27.1 kg/m²) and you are worried about missing maturity onset diabetes of the young (MODY) or type 1 diabetes. Which one of the following is most suggestive of MODY?

- ○ Ketosis during periods of hyperglycaemia ×
- ○ Family history of early onset diabetes mellitus ×
- ○ A history of polycystic ovarian syndrome ×
- ○ Lack of response to sulphonylureas ×
- ○ A history of autoimmune disease ×

Submit answer

Reference ranges

Score: **44.4%**

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46	-

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Question 46 of 217

A 36-year-old woman is reviewed 3 months post-partum. During the pregnancy she was diagnosed with gestational diabetes. Following the delivery her glycaemic control has failed to improve and she has been diagnosed as having type 2 diabetes mellitus. She is only slightly overweight (body mass index 27.1 kg/m²) and you are worried about missing maturity onset diabetes of the young (MODY) or type 1 diabetes. Which one of the following is most suggestive of MODY?

○ Ketosis during periods of hyperglycaemia ×
○ Family history of early onset diabetes mellitus ×
○ A history of polycystic ovarian syndrome ×
○ Lack of response to sulphonylureas ×
○ A history of autoimmune disease ×

Submit answer

Reference ranges

Score: **44.4%**

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A 36-year-old woman is reviewed 3 months post-partum. During the pregnancy she was diagnosed with gestational diabetes. Following the delivery her glycaemic control has failed to improve and she has been diagnosed as having type 2 diabetes mellitus. She is only slightly overweight (body mass index 27.1 kg/m²) and you are worried about missing maturity onset diabetes of the young (MODY) or type 1 diabetes. Which one of the following is most suggestive of MODY?

Ketosis during periods of hyperglycaemia	4%
Family history of early onset diabetes mellitus	79%
A history of polycystic ovarian syndrome	1%
Lack of response to sulphonylureas	9%
A history of autoimmune disease	7%

MODY is inherited in an autosomal dominant fashion so a family history is often present

Important for me Less important

The correct answer is a **family history of early onset diabetes mellitus**. Maturity onset diabetes of the young (MODY) is a group of monogenic disorders characterized by autosomal dominant inheritance, early onset of hyperglycemia (usually before 25 years of age), and a primary defect in pancreatic beta-cell function. A family history of early onset diabetes mellitus is most suggestive of MODY because it indicates that there may be a genetic predisposition to the condition, which is consistent with the autosomal dominant inheritance pattern seen in MODY.

Ketosis during periods of hyperglycaemia is more suggestive of type 1 diabetes rather than MODY. Type 1 diabetes results from autoimmune destruction of pancreatic beta cells, leading to an absolute insulin deficiency. In the absence of sufficient insulin, the body relies on breaking down fats for energy, resulting in ketone production and ketosis. In contrast, patients with MODY typically have a defect in pancreatic beta-cell function but still produce some insulin, so they are less likely to develop ketosis.

A **history of polycystic ovarian syndrome** (PCOS) is not particularly suggestive of MODY. PCOS is associated with insulin resistance and an increased risk for developing type 2 diabetes mellitus. While some forms of MODY can be associated with insulin resistance, it would not be considered the most characteristic feature when trying to differentiate between types of diabetes.

Lack of response to sulphonylureas can be seen in some subtypes of MODY (e.g., MODY 1 and MODY 3). However, it does not necessarily rule out other types or subtypes since there are several different genetic defects that can cause MODY. Furthermore, lack of response to sulphonylureas could also occur in type 2 diabetics who have progressed to needing insulin therapy.

A **history of autoimmune disease** is more suggestive of type 1 diabetes, as it results from an autoimmune process that destroys pancreatic beta cells. While there is no strong association between MODY and autoimmune diseases, a history of autoimmune disease could increase the likelihood of developing other types of diabetes, such as type 1 or Latent Autoimmune Diabetes in Adults (LADA).

👍 👎 Discuss (5) Improve

Next question

MODY ★

Maturity-Onset Diabetes of the Young (MODY) is a form of monogenic diabetes, typically characterized by an autosomal dominant inheritance pattern, onset usually before 25 years of age, and impairment in insulin secretion with minimal or no defects in insulin action. Unlike more common forms of diabetes, such as Type 1 and Type 2, MODY is not primarily driven by lifestyle factors.

It is thought that around 1-2% of patients with diabetes mellitus have MODY, and around 90% are misclassified as having either type 1 or type 2 diabetes mellitus.

Classification and Genetics

MODY is a heterogenous group, with over 14 types identified as of the latest revision, each corresponding to mutations in different genes. The most common types are MODY2 (GCK mutation) and MODY3 (HNF1A mutation), but others include MODY1 (HNF4A), MODY4 (PDX1), MODY5 (HNF1B), and so on.

MODY 3 accounts for around 60% of cases with MODY 2 causing 20% of cases.

Each subtype varies in its clinical presentation, management, and prognosis, emphasizing the importance of precise genetic diagnosis in order to guide treatment strategy and genetic counselling.

Clinical Features

Patients with MODY often present with mild non-ketotic hyperglycemia that is often detected incidentally or during routine screening. It may also be discovered during pregnancy. Unlike Type 1 diabetes, patients with MODY usually do not present with diabetic ketoacidosis except under severe stress conditions, and unlike Type 2 diabetes, they are often of normal weight and do not exhibit signs of insulin resistance.

The specific clinical manifestations and complications can vary depending on the subtype of MODY. For example, individuals with MODY2 generally have mild, stable fasting hyperglycemia and rarely develop severe complications, whereas those with MODY3 or MODY1 may have progressive hyperglycemia and are at higher risk for complications typically associated with diabetes, such as retinopathy, nephropathy, and cardiovascular disease.

Diagnosis

MODY should be suspected in individuals with persistent, asymptomatic hyperglycemia detected before the age of 25, without the typical features of Type 1 or Type 2 diabetes.

The diagnosis is confirmed by genetic testing, which is crucial for identifying the specific type of MODY, as this has direct implications for management. It's important to remember, however, that genetic testing can be expensive and may not be available in all settings.

Treatment

The treatment for MODY depends on the specific genetic subtype. For example, MODY2 often does not require specific treatment, as the hyperglycemia is mild and usually does not lead to complications. In contrast, MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas. Insulin therapy may be necessary in some cases, especially during pregnancy or if sulfonylureas are contraindicated or ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **45.7%**

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A 72-year-old woman is recovering on the neurosurgical unit following a subdural haemorrhage. Four days earlier she underwent Burr hole surgery. You are asked to see her due to a persistently low sodium for the past three days. You note the following investigations:

Day 2 post-surgery

Serum Na+	116 mmol/l

Day 3 post-surgery

Serum Na+	117 mmol/l

Day 4 post-surgery

Serum Na+	115 mmol/l
Urinary Na+	25 mmol/l
Serum osmolality	280 mmol/l

Examination of the patient demonstrates dry mucous membranes and delayed capillary refill time.

What is the most likely diagnosis?

- ○ SIADH ×
- ○ Diabetes insipidus ×
- ○ Cerebral salt wasting syndrome ×
- ○ Renal tubular acidosis type IV ×
- ○ Sheehan's syndrome ×

Submit answer

Reference ranges ∨

Score: **45.7%**

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Day 3 post-surgery

Serum Na+	117 mmol/l

Day 4 post-surgery

Serum Na+	115 mmol/l
Urinary Na+	25 mmol/l
Serum osmolality	280 mmol/l

Examination of the patient demonstrates dry mucous membranes and delayed capillary refill time.

What is the most likely diagnosis?

	SIADH	34%
	Diabetes insipidus	12%
🟩	Cerebral salt wasting syndrome	51%
	Renal tubular acidosis type IV	1%
🟥	Sheehan's syndrome	2%

Diabetes insipidus is classically associated with hypernatraemia. Sheehan's syndrome refers to the specific situation of pituitary necrosis following childbirth. The cardinal feature of renal tubular acidosis type IV is hyperkalaemia.

This leaves SIADH and cerebral salt wasting syndrome. The hydration status in this patient can be considered hypovolaemic making SIADH unlikely (typically euvolaemic). Additionally, this diagnosis should only be made in the absence of hypothyroidism and adrenal dysfunction.

Cerebral salt wasting syndrome can occur following neurosurgery. It occurs due to sodium wasting in the urine. Comparatively, it is treated with replacing fluid and sodium losses.

👍 👎 Discuss (6) Improve

Next question

Hyponatraemia ★

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)

- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic

- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss

- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

+ Q 123

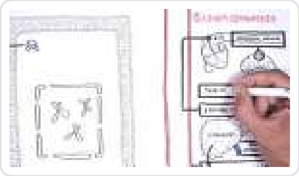
Next question

Textbooks

High-yield textbook

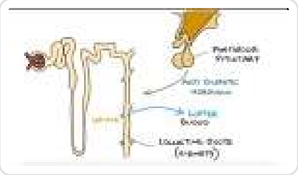
Extended textbook

Media



Hyponatraemia - classification, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 5 👎 0



Understanding Hyponatraemia

Zero To Finals - YouTube 👍 17 👎 1

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **44.7%**

1 ✗
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Question 48 of 217

A 68-year-old Indian patient presents to the emergency department with facial tetany, muscle cramps and paraesthesia of her fingers and toes. This is her second admission with similar symptoms. Her past medical history includes diffuse cutaneous systemic sclerosis with gastrointestinal, cutaneous and pulmonary manifestations. She was also diagnosed with vitamin D deficiency two years ago and receives regular vitamin D supplements. Her blood tests are as follows:

Hb	124 g/l
WBC	8.0 * 10^9/l
Na^+	141 mmol/l
K^+	4.3 mmol/l
Urea	6.5 mmol/l
Creatinine	90 µmol/l
CRP	15 mg/l
Corrected calcium	1.68 mmol/l
Phosphate	1.4 mmol/l
Magnesium	0.28 mmol/l
PTH	2 pmol/L (normal range = 8.5-12)
Amylase	14 u/l

Her symptoms improve with intravenous calcium replacement and intravenous magnesium replacement, correcting both electrolytes to within normal range. What is the underlying cause for these metabolic disturbances in this patient?

○ Hypomagnesaemia ×

○ Primary hypoparathyroidism ×

○ Insufficient vitamin D supplementation ×

○ Chronic kidney failure ×

○ Chronic pancreatitis ×

Submit answer

Reference ranges ⌄

Score: **44.7%**

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Question 48 of 217 ✓

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Amylase	14 u/l

Her symptoms improve with intravenous calcium replacement and intravenous magnesium replacement, correcting both electrolytes to within normal range. What is the underlying cause for these metabolic disturbances in this patient?

Hypomagnesaemia	28%
Primary hypoparathyroidism	58%

Insufficient vitamin D supplementation	9%
Chronic kidney failure	3%
Chronic pancreatitis	2%

Magnesium deficiency causes hypocalcaemia

Important for me Less important

This complex picture investigates the underlying cause of hypomagnesaemia and hypocalcaemia in a patient with significant GI disease. With regular vitamin D supplementation, it is unlikely this is the cause. Her renal function is also within normal range. Although her parathyroid hormone levels are low, the likely underlying cause is due to insufficient magnesium absorption due to GI systemic sclerosis, which results in reduces parathyroid hormone release. There is nothing in the history to suggest a primary hypoparathyroidism or chronic pancreatitis.

👍 👎 Discuss (26) Improve

Next question

Hypocalcaemia: causes and management

The clinical history combined with parathyroid hormone levels will reveal the cause of hypocalcaemia in the majority of cases

Causes
- vitamin D deficiency (osteomalacia)
- chronic kidney disease
- hypoparathyroidism (e.g. post thyroid/parathyroid surgery)
- pseudohypoparathyroidism (target cells insensitive to PTH)
- rhabdomyolysis (initial stages)
- magnesium deficiency (due to end organ PTH resistance)
- massive blood transfusion
- acute pancreatitis

Contamination of blood samples with EDTA may also give falsely low calcium levels.

Management

- severe hypocalcaemia (e.g. carpopedal spasm, tetany, seizures or prolonged QT interval) requires IV calcium replacement
    - the preferred method is with intravenous calcium gluconate, 10ml of 10% solution over 10 minutes
    - intravenous calcium chloride is more likely to cause local irritation
    - ECG monitoring is recommended
- further management depends on the underlying cause

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 3 👎 2

Report broken media

Score: **45.8%**

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A 34-year-old patient is brought by ambulance to the emergency department with a decreased level of consciousness. She has a past medical history of type 2 diabetes and emotionally unstable personality disorder. She takes metformin and gliclazide. She smokes ten cigarettes daily. She is unemployed.

Her observations are heart rate 111 beats per minute, blood pressure 101/55 mmHg, respiratory rate 21/minute, oxygen saturations 96% on room air and temperature 37°C.

On examination, her Glasgow coma scale is 9/15 (E1 V3 M5). The cardio-respiratory examination is normal. Abdominal examination is normal. Neurological examination reveals normal tone, downgoing plantars. Her pupils are equal and reactive to light.

A bedside blood glucose level is 1.8 mmol/L.

Despite 2 x 100ml boluses of 10% dextrose and a subsequent dextrose infusion, the patient experiences recurrent hypoglycaemia.

What is the next most appropriate pharmacological treatment?

- ○ Activated charcoal ×
- ○ Enteral supplementation ×
- ○ Octreotide ×
- ○ 10% dextrose ×
- ○ 50% dextrose ×

Submit answer

Reference ranges

Score: **45.8%**

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- ○ Enteral supplementation ×
- ● Octreotide ×
- ○ 10% dextrose ×
- ○ 50% dextrose ×

Submit answer

Reference ranges

Score: **45.8%**

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A bedside blood glucose level is 1.8 mmol/L.

Despite 2 x 100ml boluses of 10% dextrose and a subsequent dextrose infusion, the patient experiences recurrent hypoglycaemia.

What is the next most appropriate pharmacological treatment?

Activated charcoal	2%
Enteral supplementation	1%
[✓ Octreotide]	88%
10% dextrose	1%
50% dextrose	8%

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Important for me Less important

Octreotide is correct. This patient has recurrent hypoglycaemia in the context of known sulphonylurea use an emotionally unstable personality disorder. It is likely that she has taken a sulphonylurea overdose. Despite dextrose supplementation, she experiences recurrent hypoglycaemia. Octreotide blocks insulin secretion. The rationale being that dextrose precipitates further insulin release, causing a vicious cycle of insulin secretion. Octreotide prevents further insulin release in sulphonylurea overdose and is an option if the patient remains hypoglycaemic.

Activated charcoal is incorrect. This typically needs to be given within 1-2 hours of administration of sulphonylureas and we do not know the time of ingestion.

10% dextrose is incorrect. Despite sufficient dextrose administration, the patient remains hypoglycaemic and therefore an alternative treatment is needed.

Enteral supplementation is incorrect. The patient has a GCS of 9/15 and therefore enteral supplementation would be inappropriate.

50% dextrose is incorrect. The patient has already had a typically sufficient administration of dextrose. The patient remains hypoglycaemic, likely because of ongoing endogenous secretion of insulin in response to the glucose load and needs alternative treatment that blocks insulin secretion.

👍 👎 Discuss (4) Improve

Next question

Hypoglycaemia ★

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas

- liver failure
- Addison's disease
- alcohol
    - causes exaggerated insulin secretion
    - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia

- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later
- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic) neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features

- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
    - Sweating
    - Shaking
    - Hunger
    - Anxiety
    - Nausea
- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
    - Weakness
    - Vision changes
    - Confusion
    - Dizziness
- Severe and uncommon features of hypoglycaemia include:
    - Convulsion

    - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
    - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form
    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:

  - If the patient is alert, a quick-acting carbohydrate may be given (as above)
  - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
  - Alternatively, intravenous 20% glucose solution may be given through a large vein

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **46.9%**

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Question 50 of 217

A 66-year-old male was admitted with agitation and confusion, worsening over the past 1 week. His past medical history includes hypertension, ischaemic heart disease and chronic back pain. His daughter noticed that he had lost about 1 stone in weight (currently weighs 71 kg), has been more tired over the last month and that he has been drinking a lot more water. This was associated with the development of urinary incontinence.

On examination, his heart rate was 108 beats/min, blood pressure was 95/42 mmHg, saturations were 94% on air and respiratory rate was 20/min. He is confused, with a Glasgow Coma Scale of 14 and appeared dehydrated.

Blood results are as follows:

Na^+	125 mmol/l
K^+	5.0 mmol/l
Urea	18 mmol/l
Creatinine	180 µmol/l
Blood glucose	34 mmol/l

Venous blood gas was done and showed the following:

pH	7.32
pCO2	4.6 kPa
pO2	6.1 kPa
HCO3	17mmol/l
BE	-3.6 mmol/l

Which is the most important treatment?

- ○ Intravenous 0.9% sodium chloride ✕
- ○ 10 units of human actrapid stat ✕
- ● Start insulin sliding scale at 6 units/hr ✕
- ○ Calcium gluconate ✕
- ○ Intravenous 1.8% sodium chloride ✕

Submit answer

Reference ranges

Score: **46.9%**

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pH	7.32
pCO2	4.6 kPa
pO2	6.1 kPa
HCO3	17mmol/l
BE	-3.6 mmol/l

Which is the most important treatment?

Intravenous 0.9% sodium chloride	92%
10 units of human actrapid stat	1%
Start insulin sliding scale at 6 units/hr	2%
Calcium gluconate	1%
Intravenous 1.8% sodium chloride	4%

Characteristic features of hyperosmolar hyperglycaemic state (HHS) includes:

- high osmolality, often 320 mosmol/kg or more
- high blood glucose, usually 30 mmol/L or more
- severely dehydrated and unwell.
- without significant hyperketonaemia or acidosis

HHS typically occurs in the elderly and is often the first presentation of Type 2 Diabetes Mellitus.

Using the blood results, osmolality can be calculated with the formula 2(Na+K) +glucose+urea

Goals of treatment include:

- Normalise the osmolality
- Replace fluid and electrolyte losses
- Normalise blood glucose
- Prevention of complications: Arterial or venous thrombosis/cerebral oedema

Fluid replacement must commence first; an initial insulin bolus of 0.15 U per kg may be given once infusions are underway. Fluid replacement alone with 0.9% sodium chloride solution will result in falling blood glucose. Insulin treatment prior to adequate fluid replacement may result in cardiovascular collapse as water moves out of the intravascular space, with a resulting decline in intravascular volume.

Patients with HHS are often exquisitely sensitive to insulin and require much lower doses than in diabetic ketoacidosis (DKA).The recommended insulin dose is a fixed rate intravenous insulin infusion (FRIII) given at 0.05 units per kg per hour (e.g. 4 units/hr in an 80 kg man) is used

Beware of rapid correction of hyponatraemia, may lead to cerebral pontine myelinolysis

Source:
http://www.diabetes.org.uk/Documents/Position%20statements/JBDS-IP-HHS-Adults.pdf

👍 👎 Discuss (9) Improve

Next question

Hyperosmolar hyperglycaemic state ★

Hyperosmolar hyperglycaemic state (HHS) is a medical emergency that can be difficult to manage and has a significant associated mortality (up to 20%). Hyperglycaemia results in osmotic diuresis, severe dehydration, and electrolyte deficiencies. HHS typically presents in the elderly with type 2 diabetes mellitus (T2DM).

Pathophysiology

- hyperglycaemia → ↑ serum osmolality → osmotic diuresis → severe volume depletion

Precipitating factors

- intercurrent illness
- dementia
- sedative drugs

Clinical features

- whilst DKA presents within hours of onset, HHS comes on over many days, and consequently, the dehydration and metabolic disturbances may be more extreme
- consequences of volume loss
    - clinical signs of dehydration
    - polyuria
    - polydipsia
- systemic
    - lethargy
    - nausea and vomiting
- neurological
    - altered level of consciousness
    - focal neurological deficits
- haematological
    - hyperviscosity (may result in myocardial infarctions, stroke and peripheral arterial thrombosis)

There are no precise **diagnostic criteria** but the following are typically seen

- hypovolaemia
- marked hyperglycaemia (>30 mmol/L)
- significantly raised serum osmolarity (> 320 mosmol/kg)
    - can be calculated by: 2 * Na^+ + glucose + urea
- no significant hyperketonaemia (<3 mmol/L)
- no significant acidosis (bicarbonate > 15 mmol/l or pH > 7.3 - acidosis can occur due to lactic acidosis or renal impairment)

Management

- fluid replacement
    - fluid losses in HHS are estimated to be between 100 - 220 ml/kg
    - IV 0.9% sodium chloride solution
    - typically given at 0.5 - 1 L/hour depending on clinical assessment
    - potassium levels should be monitored and added to fluids depending on the level
- insulin

    - should not be given unless blood glucose stops falling while giving IV fluids
- venous thromboembolism prophylaxis
    - patients are at risk of thrombosis due to hyperviscosity

Complications

- vascular complications may occur due to hyperviscosity:
    - such as myocardial infarction
    - stroke

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Diabetes Societies 👍 20 👎 13

The management of the hyperosmolar hyperglycaemic state (HHS)

Suggest link Report broken link

Score: **48%**

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A 28-year-old pregnant lady presents to the Emergency Department with palpitations and sweating. She mentions that she has had these symptoms on and off for the past 4 months but that they have worsened over the past few weeks. Now she is feeling worried and wanted to be assessed medically due to her concern she was having a miscarriage. She looks particularly anxious to be in hospital. This is her first pregnancy. She is 7 weeks pregnant. She has had no vaginal bleeding or discharge during the course of her pregnancy. She is normally fit and well.

Initial observations reveal a blood pressure of 130/85 mmHg, a heart rate of 110 beats per minute, a respiratory rate of 19/min, oxygen saturations of 99% on air and a temperature of 37.5ºC. Examination findings reveal a resting tachycardia and a subtle goitre is noted.

Blood test results are as follows:

Hb	110 g/l
Wcc	12 x109/l
Plt	245 x109/l
CRP	12 mg/l
Na+	140 mmol/l
K+	5.0 mmol/l
Ur	5.7 mmol/l
Cr	110 µmol/l
D-dimer	490 ng/ml
T4	21 mU/l
TSH	<0.05 pmol/l

Given the most likely diagnosis, how should this lady be managed?

○ Watch and wait/symptomatic control with beta blockade ×

○ Radioactive iodine therapy ×

○ Subtotal thyroidectomy ×

○ Propylthiouracil ×

● Block and replace carbimazole + thyroxine ×

Submit answer

Reference ranges ∨

Score: **48%**

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Ur	5.7 mmol/l
Cr	110 µmol/l
D-dimer	490 ng/ml
T4	21 mU/l
TSH	<0.05 pmol/l

Given the most likely diagnosis, how should this lady be managed?

Watch and wait/symptomatic control with beta blockade	14%
Radioactive iodine therapy	1%
Subtotal thyroidectomy	1%
Propylthiouracil	82%
Block and replace carbimazole + thyroxine	2%

This lady has symptoms and biochemical evidence of hyperthyroidism. This lady's symptoms predate her pregnancy, therefore it is not pregnancy induced thyrotoxicosis and will not self-limit - she will need treatment to prevent complications to her and the foetus. Radioactive iodine is contraindicated. Subtotal thyroidectomy is a little risky and extreme during pregnancy. Carbimazole, whilst normally first line, has been associated with neonatal aplasia cutis before 12 weeks gestation and is therefore usually avoided. This leaves propylthiouracil as the current safest option. During the second trimester, propylthiouracil should be changed to carbimazole due to the potential risk of hepatotoxicity with propylthiouracil. The lowest dose that controls the hyperthyroid state should be used as both medications can cross the placenta.

👍 👎 Discuss (9) Improve

Next question

Pregnancy: thyroid problems ★

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link

Report broken link

Score: **49%**

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A 38-year-old woman is 10 weeks pregnant and presents with increasing fatigue, weight gain and feeling very cold. Moreover, her main issue is constipation only responsive to large doses of laxatives. In recent years she has had well-controlled hypothyroidism on 50 micrograms of levothyroxine.

On examination her hair is very thin, her skin feels slightly waxy, she has mild peripheral oedema and has a larger body habitus than previously.

Blood tests are as follows:

Calcium	2.4 mmol/L	(2.1-2.6)
Phosphate	1.1 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	5.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	9.8 pmol/L	(9.0 - 18)

What is the best next management option?

- ○ 100 micrograms levothyroxine ✕
- ○ 25 micrograms levothyroxine ✕
- ○ 75 micrograms levothyroxine ✕
- ○ Continue 50 micrograms levothyroxine ✕
- ○ Stop the levothyroxine ✕

Submit answer

Reference ranges ∨

Score: **49%**

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Magnesium	0.8 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	5.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	9.8 pmol/L	(9.0 - 18)

What is the best next management option?

100 micrograms levothyroxine	25%
25 micrograms levothyroxine	3%
[✓ 75 micrograms levothyroxine]	69%
Continue 50 micrograms levothyroxine	3%
Stop the levothyroxine	0%

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

Important for me Less important

75 micrograms levothyroxine is the correct answer. This is because during pregnancy (as early as 4-6 weeks) the amount of thyroglobulin binding protein increases, thus there is less free thyroid hormone in the bloodstream exacerbating this woman's state of hypothyroidism. Increasing the dose of 50 by 50% to 75 is usually sufficient as a measure to induce a state of euthyroid.

25 micrograms levothyroxine is incorrect as this would worsen the state of hypothyroidism.

Continue 50 micrograms levothyroxine is incorrect as this would not address the issue of the state of hypothyroidism on the current dose.

Stop the levothyroxine is incorrect as this would almost certainly exacerbate the state of hypothyroidism yet further.

100 micrograms levothyroxine is incorrect as this would be an increase on the dose by 100%; at this stage most likely too much and may cause a state of hyperthyroidism.

👍 👎 Discuss (2) Improve

Next question

Pregnancy: thyroid problems ★

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+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link Report broken link

Score: **50%**

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← Question 53 of 217 →

A 24-year-old female presents to the clinic with worsening of fatigue. She states that she has been feeling tired for months. On systematic enquiry she complains of constipation, cold intolerance and dry skin. She has no past medical history of note and is not on any regular medicines. On examination you note that her lying blood pressure is 110/75 mmHg which falls to 95/60 mmHg on standing.

Investigation results are as follows:

Hb	110 g/l	Na$^+$	131 mmol/l	Fasting glucose	7.5 mmol/l
Platelets	425 * 10^9/l	K$^+$	5.1 mmol/l	Bicarbonate	19 mmol/l
WBC	10.1 * 10^9/l	Urea	10.5 mmol/l	Creatinine	110 μmol/l

What is the most likely diagnosis?

- ○ Autoimmune polyendocrinopathy syndrome (APS) - type 1 ×
- ○ Autoimmune polyendocrinopathy syndrome (APS) - type 2 ×
- ○ Addison's disease ×
- ○ Type 1 diabetes mellitus ×
- ○ Type 2 diabetes mellitus ×

Submit answer

Reference ranges

Score: **50%**

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Question 53 of 217

A 24-year-old female presents to the clinic with worsening of fatigue. She states that she has been feeling tired for months. On systematic enquiry she complains of constipation, cold intolerance and dry skin. She has no past medical history of note and is not on any regular medicines. On examination you note that her lying blood pressure is 110/75 mmHg which falls to 95/60 mmHg on standing.

Investigation results are as follows:

Hb	110 g/l	Na$^+$	131 mmol/l	Fasting glucose	7.5 mmol/l
Platelets	425 * 10^9/l	K$^+$	5.1 mmol/l	Bicarbonate	19 mmol/l
WBC	10.1 * 10^9/l	Urea	10.5 mmol/l	Creatinine	110 µmol/l

What is the most likely diagnosis?

- ○ Autoimmune polyendocrinopathy syndrome (APS) - type 1 ×
- ○ Autoimmune polyendocrinopathy syndrome (APS) - type 2 ×
- ○ Addison's disease ×
- ○ Type 1 diabetes mellitus ×
- ○ Type 2 diabetes mellitus ×

Submit answer

Reference ranges

Score: **50%**

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Question 53 of 217 ✗

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Investigation results are as follows:

Hb	110 g/l	Na$^+$	131 mmol/l	Fasting glucose	7.5 mmol/l
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WBC	10.1 * 10^9/l	Urea	10.5 mmol/l	Creatinine	110 µmol/l

What is the most likely diagnosis?

🟥	Autoimmune polyendocrinopathy syndrome (APS) - type 1	38%
🟩	Autoimmune polyendocrinopathy syndrome (APS) - type 2	44%
	Addison's disease	17%
	Type 1 diabetes mellitus	1%
	Type 2 diabetes mellitus	0%

APS type 2 patients have Addison's disease plus either T1DM or autoimmune thyroid disease

Important for me Less important

A fasting glucose of 7.5 mmol/l suggests a diagnosis of diabetes. In the absence of diabetic symptoms (e.g. polyuria, and polydipsia), this would need to be repeated to confirm the diagnosis. Alternatively, other tests such as HbA1c, or an oral glucose tolerance test could be used.

The clinical features of constipation, cold intolerance and dry skin are suggestive of hypothyroidism. Thyroid function tests should be performed.

The biochemistry (hyponatraemia, hyperkalemia, and acidosis) and postural hypotension are suggestive of hypoaldosteronism.

The unifying diagnosis is autoimmune polyendocrinopathy syndrome (APS) type 2. APS type 2 patients have Addison's disease plus either T1DM and/or autoimmune thyroid disease.

👍 👎 Discuss (4) Improve

Next question

Autoimmune polyendocrinopathy syndrome

Addison's disease (autoimmune hypoadrenalism) is associated with other endocrine deficiencies in approximately 10% of patients. There are two distinct types of autoimmune polyendocrinopathy syndrome (APS), with type 2 (sometimes referred to as Schmidt's syndrome) being much more common.

APS type 2 has a polygenic inheritance and is linked to HLA DR3/DR4. Patients have Addison's disease plus either:
- type 1 diabetes mellitus
- autoimmune thyroid disease

APS type 1 is occasionally referred to as Multiple Endocrine Deficiency Autoimmune Candidiasis (MEDAC). It is a very rare autosomal recessive disorder caused by mutation of AIRE1 gene on chromosome 21

Features of APS type 1 (2 out of 3 needed)
- chronic mucocutaneous candidiasis (typically first feature as young child)

- Addison's disease
- primary hypoparathyroidism

Vitiligo can occur in both types

+ Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Media

AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE 1

Autoimmune polyglandular syndrome type 1

Osmosis - YouTube 👍 5 👎 0

Report broken media

Score: **49.1%**

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Question 54 of 217

A 64-year-old man has been admitted with hypercalcaemia on a background of metastatic prostate cancer. A recent CT of the chest, abdomen, and pelvis confirmed multiple bony, sclerotic metastases in the lumbar spine and pelvis.

IV fluids have been given since admission. IV pamidronate 60mg was given 3 days ago and IV denosumab 120mg was given yesterday.

A trend of adjusted calcium is shown below.

	Day 1	Day 2	Day 3	Today	Range
Adjusted calcium	3.32 mmol/L	3.26 mmol/L	3.24 mmol/L	3.16 mmol/L	(2.1-2.6)

The renal function today is shown below.

Na^+	126 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	132 μmol/L	(55 - 120)

What is the most appropriate next step in management?

- ○ Calcitonin ✕
- ○ Further dose of denosumab ✕
- ○ Further dose of pamidronate ✕
- ○ Indapamide ✕
- ○ Prednisolone ✕

Submit answer

Reference ranges

Score: **49.1%**

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Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	132 µmol/L	(55 - 120)

What is the most appropriate next step in management?

○ Calcitonin ×

○ Further dose of denosumab ×

○ Further dose of pamidronate ×

○ Indapamide ×

○ Prednisolone ×

Submit answer

Reference ranges ⌄

Score: **49.1%**

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Question 54 of 217 ✓

A 64-year-old man has been admitted with hypercalcaemia on a background of metastatic prostate cancer. A recent CT of the chest, abdomen, and pelvis confirmed multiple bony, sclerotic metastases in the lumbar spine and pelvis.

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Bicarbonate	23 mmol/L	(22 - 29)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	132 µmol/L	(55 - 120)

What is the most appropriate next step in management?

Calcitonin	70%
Further dose of denosumab	8%
Further dose of pamidronate	7%
Indapamide	5%
Prednisolone	9%

Refractory hypercalcaemia of malignancy may be treated with subcutaneous calcitonin if therapy with fluids and pamidronate fails

Important for me Less important

Calcitonin is the correct answer. Calcitonin has been found to be most useful when combined with hydration, bisphosphonates and/or denosumab, all of which the above patient has received. Calcitonin acts by increasing the excretion of renal calcium and decreasing bone resorption.

Denosumab is commonly used in malignancy-associated hypercalcaemia and can be used in the emergency management of refractory hypercalcaemia. Denosumab can be repeated weekly, therefore given that it was given only the day previously, it is not appropriate to give a **further dose of denosumab**.

A **further dose of pamidronate** is inappropriate as the last dose was given 3 days ago. Bisphosphonates take 2-3 days to see an effect and maximal effect may not show until the 7th day.

Loops diuretics can be used in hypercalcaemia, however, thiazide-like diuretics, such as **indapamide**, are contraindicated as they can worsen hypercalcaemia.

Steroids, such as **prednisolone**, are not beneficial for hypercalcaemia unless associated with sarcoidosis.

👍 👎 Discuss (4) Improve

Next question

Hypercalcaemia: management ★

The initial management of hypercalcaemia is rehydration with normal saline, typically 3-4 litres/day. Following rehydration bisphosphonates may be used. They typically take 2-3 days to work with maximal effect being seen at 7 days

Other options include:
- calcitonin - quicker effect than bisphosphonates
- steroids in sarcoidosis

Loop diuretics such as furosemide are sometimes used in hypercalcaemia, particularly in patients who cannot tolerate aggressive fluid rehydration. However, they should be used with caution as they may worsen electrolyte derangement and volume depletion.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 4 👎 7

Hypercalcaemia â€“ presentation and management

Suggest link Report broken link

Media

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 0 👎 1

Report broken media

Score: **50%**

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A 22-year-old man presents with excessive thirst and urination. He has a strong family history of diabetes. His BMI is 20.5 kg/m².

Blood results are as follows:

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	210 * 10^9/L	(150 - 400)
WBC	10.8 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	74 μmol/L	(55 - 120)
Random glucose	14.1 mmol/l	(<11.1)
CRP	2 mg/L	(< 5)

Which mutation is associated with the best response to low-dose sulphonylureas?

- ○ Glucokinase ×
- ○ HNF1A mutation ×
- ○ HNF1B mutation ×
- ○ HNF4A mutation ×
- ○ PDX1 mutation ×

Submit answer

Reference ranges

Score: **50%**

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Question 55 of 217 ✗

A 22-year-old man presents with excessive thirst and urination. He has a strong family history of diabetes. His BMI is 20.5 kg/m².

Blood results are as follows:

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	210 * 10^9/L	(150 - 400)
WBC	10.8 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
Random glucose	14.1 mmol/l	(<11.1)
CRP	2 mg/L	(< 5)

Which mutation is associated with the best response to low-dose sulphonylureas?

	Glucokinase	8%
🟩	HNF1A mutation	54%
🟥	HNF1B mutation	16%
	HNF4A mutation	21%
	PDX1 mutation	2%

MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

Important for me Less important

The patient most likely has a diagnosis of maturity-onset diabetes of the young (MODY) given his raised blood glucose, young age, strong family history of diabetes, and normal BMI.

HNF1A mutation is correct. MODY associated with HNF1A often responds well to treatment with low-dose sulfonylureas.

Glucokinase is incorrect. This mutation is associated with MODY 2. This subtype has a low rate of complications and often does not need treatment.

HNF1B mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

HNF4A mutation is incorrect. Although patients with this subtype may also respond favourably to sulphonylureas, the response is usually less marked compared to HNF1A mutations.

PDX1 mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

👍 👎 Discuss (1) Improve

Next question

MODY ★

Maturity-Onset Diabetes of the Young (MODY) is a form of monogenic diabetes, typically characterized by an autosomal dominant inheritance pattern, onset usually before 25 years of age, and impairment in insulin secretion with minimal or no defects in insulin action. Unlike more common forms of diabetes, such as Type 1 and Type 2, MODY is not primarily driven by lifestyle factors.

It is thought that around 1-2% of patients with diabetes mellitus have MODY, and around 90% are misclassified as having either type 1 or type

2 diabetes mellitus.

Classification and Genetics

MODY is a heterogenous group, with over 14 types identified as of the latest revision, each corresponding to mutations in different genes. The most common types are MODY2 (GCK mutation) and MODY3 (HNF1A mutation), but others include MODY1 (HNF4A), MODY4 (PDX1), MODY5 (HNF1B), and so on.

MODY 3 accounts for around 60% of cases with MODY 2 causing 20% of cases.

Each subtype varies in its clinical presentation, management, and prognosis, emphasizing the importance of precise genetic diagnosis in order to guide treatment strategy and genetic counselling.

Clinical Features

Patients with MODY often present with mild non-ketotic hyperglycemia that is often detected incidentally or during routine screening. It may also be discovered during pregnancy. Unlike Type 1 diabetes, patients with MODY usually do not present with diabetic ketoacidosis except under severe stress conditions, and unlike Type 2 diabetes, they are often of normal weight and do not exhibit signs of insulin resistance.

The specific clinical manifestations and complications can vary depending on the subtype of MODY. For example, individuals with MODY2 generally have mild, stable fasting hyperglycemia and rarely develop severe complications, whereas those with MODY3 or MODY1 may have progressive hyperglycemia and are at higher risk for complications typically associated with diabetes, such as retinopathy, nephropathy, and cardiovascular disease.

Diagnosis

MODY should be suspected in individuals with persistent, asymptomatic hyperglycemia detected before the age of 25, without the typical features of Type 1 or Type 2 diabetes.

The diagnosis is confirmed by genetic testing, which is crucial for identifying the specific type of MODY, as this has direct implications for management. It's important to remember, however, that genetic testing can be expensive and may not be available in all settings.

Treatment

The treatment for MODY depends on the specific genetic subtype. For example, MODY2 often does not require specific treatment, as the hyperglycemia is mild and usually does not lead to complications. In contrast, MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas. Insulin therapy may be necessary in some cases, especially during pregnancy or if sulfonylureas are contraindicated or ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **49.1%**

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Question 56 of 217

A 61-year-old man with type 2 diabetes mellitus (T2DM) attends a diabetic review. He has been treated with metformin 1g twice daily for the last six months. He has been compliant with the metformin and had no side effects. An HbA1c is performed.

HbA1c	61mmol/mol	(<48)

What is the next step in management?

- ○ Add second oral agent ×
- ○ Increase metformin to three times daily ×
- ○ No change required ×
- ○ Start insulin ×
- ○ Switch metformin to modified release ×

Submit answer

Reference ranges

Score: **49.1%**

1 ✗
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Question 56 of 217 ✓

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HbA1c	61mmol/mol	(<48)

What is the next step in management?

Add second oral agent	70%
Increase metformin to three times daily	15%
No change required	12%
Start insulin	1%
Switch metformin to modified release	2%

T2DM on metformin, if HbA1c has risen to 58 mmol/mol then one of the following should be offered depending on the individual clinical scenario:

- DPP-4 inhibitor
- pioglitazone
- sulfonylurea
- SGLT-2 inhibitor (if NICE criteria met)

Important for me Less important

As this man has an HbA1c > 58mmol/mol despite taking metformin therapy for several months, **adding a second oral agent** is required to improve glycaemic control.

The maximum daily metformin dose is 2g. **Increasing the metformin dose to three times daily** is therefore inappropriate.

This patient has been on metformin for several months so the HbA1c should be reflective of his glycaemic control. As per the guidance, further optimisation of therapy is required if the HbA1c is > 58, Therefore it is incorrect to suggest that **no change is required**.

Starting insulin is only indicated once at least two oral agents have not achieved glycaemic control. As the patient is only on one agent at present, it would be premature to start insulin at this stage.

Switching metformin to modified release may help patients to tolerate the drug but would not affect glycaemic control. This is therefore not sufficient to address the high HbA1c.

👍 | 👎 | 💬 Discuss (1) | Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice
- encourage high fibre, low glycaemic index sources of carbohydrates

- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

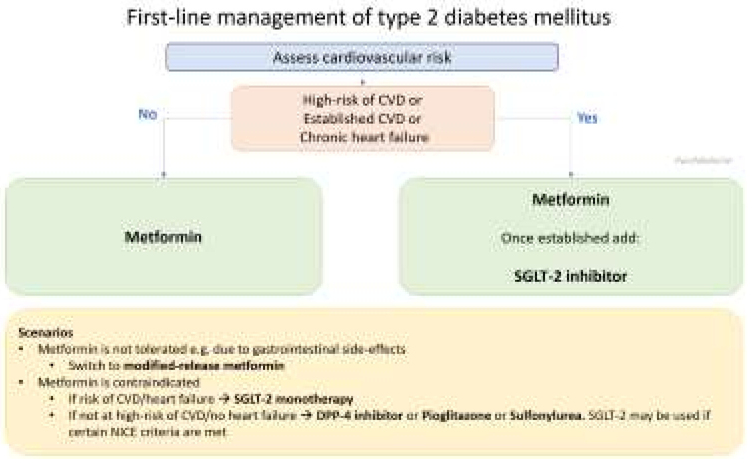
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
    - Switch to **modified-release metformin**
- Metformin is contraindicated
    - If risk of CVD/heart failure → **SGLT-2 monotherapy**
    - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

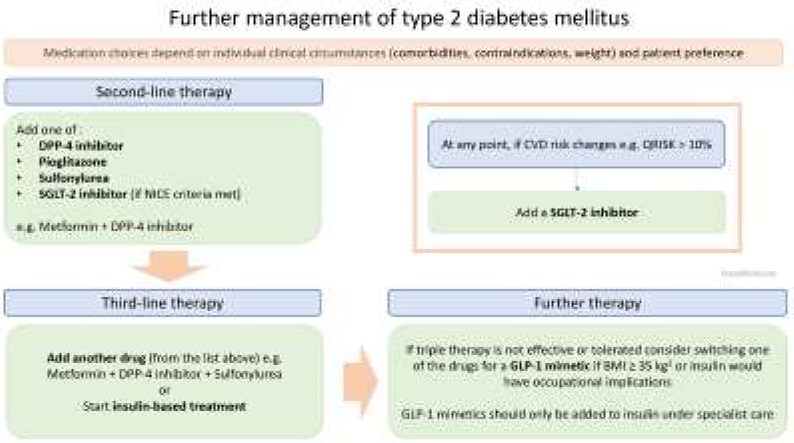
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of:
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg/m² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is

indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

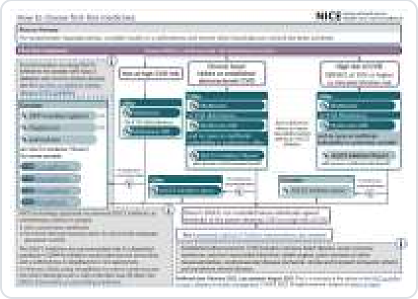
GLP-1 mimetics should only be added to insulin under specialist care

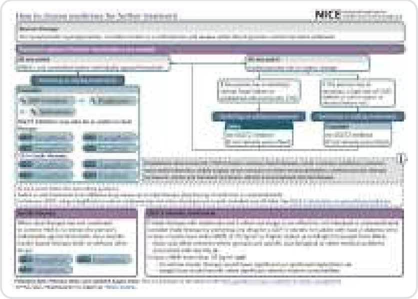
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

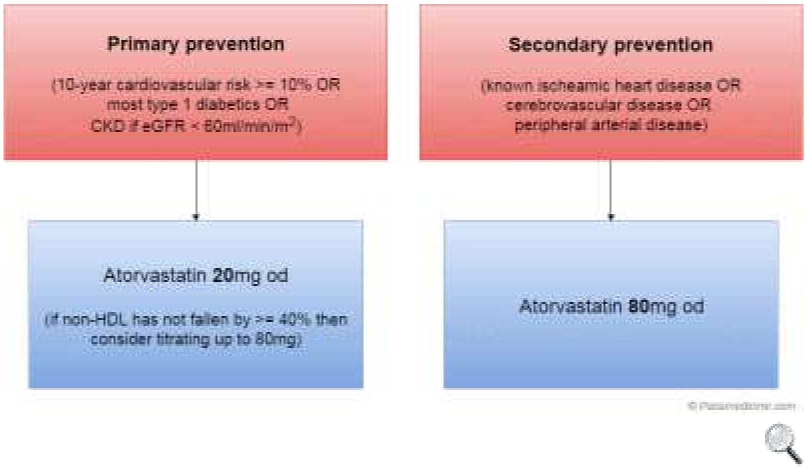
Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be

offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischaemic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

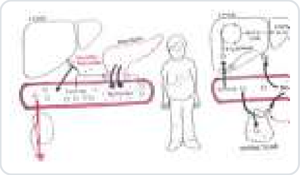
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

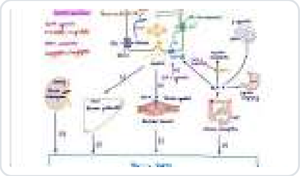
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **50%**

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Question 57 of 217

A 30-year-old south Asian woman is admitted to the accident and emergency department with abdominal pain. She is thought to be constipated. Initial blood results with subsequent tests are listed below. Urine is clear and an ECG performed is normal. Examination is unremarkable with no oedema, and blood pressure 105/68 mmHg.

pH	7.250
Bicarbonate	18.0 mmol/l
Base excess	-8.0 mmol/l
Anion gap	Normal

Potassium	7.2 mmol/l
Creatinine	56 mmol/l
Glucose	5.3 mmol/l
Thyroid function	Normal
Aldosterone	Normal
Renin	Normal
Protein electrophoresis & immunoglobulins	Normal
Urinary sodium	94 mmol/l (normal range >20 mmol/L)
Urinary potassium	26.8 mmol/l (normal range >25 mmol/L)
17- hydroxyprogesterone	Normal
Short synacthen test (basal)	320 nmol/l
Short synacthen test (30 mins)	750 nmol/l

What is the likely diagnosis?

○ Renal tubular acidosis type 1 ✕

○ Renal tubular acidosis type 2 ✕

○ Renal tubular acidosis type 4 ✕

○ Gitelman syndrome ✕

○ Adrenal insufficiency ✕

Submit answer

Reference ranges ⌄

Dr. Jamil Alrajhi

Score: 50%

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← Question 57 of 217 ✓ ⚐ →

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Urinary potassium	26.8 mmol/l (normal range >25 mmol/L)
17- hydroxyprogesterone	Normal
Short synacthen test (basal)	320 nmol/l
Short synacthen test (30 mins)	750 nmol/l

What is the likely diagnosis?

Renal tubular acidosis type 1 5%

Renal tubular acidosis type 2 5%

Renal tubular acidosis type 4	67%
Gitelman syndrome	6%
Adrenal insufficiency	17%

Renal tubular acidosis type 4 is a condition associated with increased urinary ammonia secondary to hypoaldosteronism or pseudohypoaldosteronism. This leads to hyperkalaemia and a hyperchloraemic metabolic acidosis with a normal anion gap.
Both renal tubular acidosis type 1 & 2 lead to low potassium in the context of acidosis.
Gitleman: An autosomal recessive kidney disorder characterized by hypokalemic metabolic alkalosis with hypocalciuria and hypomagnesemia.

Adrenal insufficiency is effectively ruled out with a normal short synacthen test. Candidates would be expected to understand how to perform a short synacthen test and to interpret the results. Laboratory references vary, but in general a basal plasma cortisol should exceed 170 nmol/L and should rise to above 580 nmol/L.

Discuss (6) Improve

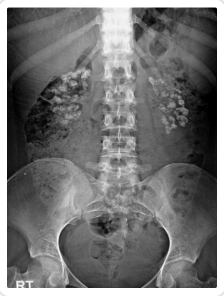
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones

- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

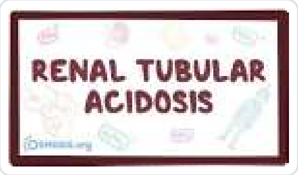
Next question

Textbooks

High-yield textbook

Extended textbook

Media


RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **50.9%**

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Question 58 of 217

A 29-year-old nulligravida woman attends her GP as she has not menstruated for 6 months. Menarche was at age 14. She had irregular periods from age 14-16, and then took oral hormonal contraception until last year when she got married. The patient does not know her family history as she is adopted. She is otherwise fit and well, enjoying running recreationally and eating healthy. She does not drink, smoke or use illicit drugs. Her vital signs are normal and body mass index is 22 kg/m².

Physical examination shows no abnormalities. Urine pregnancy test is negative.

Which of the following is the most appropriate screenshot test for this patient?

○ Karyotype ✕

○ MRI of the pituitary ✕

● ~~Serum 17-hydroxyprogesterone~~ ✕

○ Serum prolactin ✕

○ Ultrasound of the pelvis ✕

Submit answer

Reference ranges

Score: **50.9%**

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Physical examination shows no abnormalities. Urine pregnancy test is negative.

Which of the following is the most appropriate screening test for this patient?

	Karyotype	6%
	MRI of the pituitary	3%
	Serum 17-hydroxyprogesterone	21%
🟩	Serum prolactin	44%
🟥	Ultrasound of the pelvis	26%

The patients presentation is consistent with secondary amenorrhoea. The first investigative step is normally to do a pregnancy test, which in this case is negative. There are no obvious explanations for secondary amenorrhoea from her presentation. Hence the next step is to obtain serum prolactin, TSH and FSH blood results (as this will help differentiate between the most common causes of secondary amenorrhoea following pregnancy: hyperprolactinaemia, thyroid dysfunction and premature ovarian failure respectively).

Choice 1: Premature ovarian insufficiency is characterised by elevated

FSH and low oestradiol. A karyotype may be considered if Turner syndrome or fragile X syndrome is suspected, as these patients usually have ovarian dysfunction. However, this patient has no dysmorphic features or other co-morbidities to warrant karyotype evaluation.

Choice 2: An MRI of the pituitary should be performed in patients with persistently elevated prolactin (with or without galactorrhoea), unexplained headaches, bitemporal hemianopia or other concerning neurologic findings. It is not an appropriate screening test in this patient.

Choice 3: Serum 17-hydroxyprogesterone is elevated in non-classic congenital adrenal hyperplasia due to 21-hydroxylase deficiency, a condition that presents with hyperandrogenism in late childhood. This patient has no findings of androgen excess (e.g. male pattern hair distribution, severe acne).

Choice 5: An ultrasound may be indicated in patients with abnormal pelvic pain, pelvic organ enlargement or masses on examination, or signs of hyperandrogenism (e.g. polycystic ovarian syndrome, androgen tumour), none of which are seen in this patient.

👍 👎 💬 Discuss (4) Improve

Next question

Prolactin and galactorrhoea ★

Prolactin is secreted by the anterior pituitary gland with release being controlled by a wide variety of physiological factors. Dopamine acts as the primary prolactin releasing inhibitory factor and hence dopamine agonists such as bromocriptine may be used to control galactorrhoea. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia

Features of excess prolactin

- men: impotence, loss of libido, galactorrhoea
- women: amenorrhoea, galactorrhoea

Causes of raised prolactin

- prolactinoma
- pregnancy
- oestrogens
- physiological: stress, exercise, sleep
- acromegaly: 1/3 of patients
- polycystic ovarian syndrome
- primary hypothyroidism (due to thyrotrophin releasing hormone (TRH) stimulating prolactin release)

Drug causes of raised prolactin

- metoclopramide, domperidone
- phenothiazines
- haloperidol
- very rare: SSRIs, opioids

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 11 👎 5

Galactorrhoea review

Suggest link　Report broken link

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **50%**

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A 54-year-old woman was admitted following abnormal blood tests on routine monitoring. She is currently undergoing palliative chemotherapy with FOLFIRINOX, a combination of folinic acid, fluorouracil, irinotecan and oxaliplatin for metastatic pancreatic cancer. She has suffered from diarrhoea, vomiting, nausea and fatigue since starting treatment. She has a past medical history of hypothyroidism, epilepsy and bipolar disorder. Her regular medications include levothyroxine, levetiracetam, lithium, loperamide, paracetamol, oramorph, zomorph, movicol and ondansetron.

Na^+	142mmol/l
K^+	3.8mmol/l
Urea	4.4mmol/l
Creatinine	83µmol/l
Corrected calcium	3.3µmol/l

She is started on treatment with IV 0.9% saline and given 90mg of IV alendronate after further blood tests are sent for, and an ECG is done. Which of her regular medications should not be prescribed on admission?

- ○ Zomorph ×
- ○ Levetiracetam ×
- ○ Loperamide ×
- ○ Levothyroxine ×
- ○ Lithium ×

Submit answer

Reference ranges

Score: **50%**

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PassMedicine

Question 59 of 217 ✗

A 54-year-old woman was admitted following abnormal blood tests on routine monitoring. She is currently undergoing palliative chemotherapy with FOLFIRINOX, a combination of folinic acid, fluorouracil, irinotecan and oxaliplatin for metastatic pancreatic cancer. She has suffered from diarrhoea, vomiting, nausea and fatigue since starting treatment. She has a past medical history of hypothyroidism, epilepsy and bipolar disorder. Her regular medications include levothyroxine, levetiracetam, lithium, loperamide, paracetamol, oramorph, zomorph, movicol and ondansetron.

Na^+	142mmol/l
K^+	3.8mmol/l
Urea	4.4mmol/l
Creatinine	83µmol/l
Corrected calcium	3.3µmol/l

She is started on treatment with IV 0.9% saline and given 90mg of IV alendronate after further blood tests are sent for, and an ECG is done. Which of her regular medications should not be prescribed on admission?

	Zomorph	6%
🟥	Levetiracetam	4%
	Loperamide	18%
	Levothyroxine	17%
🟩	Lithium	55%

This patient has developed hypercalcaemia in the context of cancer but whilst also taking lithium. Lithium can be a cause of elevated calcium and should, therefore, be held in the acute context until the cause of hypercalcaemia is established. Note that the serum concentration of lithium does not correlate with the likelihood of it being a cause of hypercalcaemia. In the context of new confusion or dehydration, withholding the opiate medications would have been reasonable.

Source:
'Assessment of Hypercalcaemia.' BMJ Best Practice. N.p., 01 June 2015

👍 👎 Discuss (6) Improve

Next question

Hypercalcaemia: causes ★

Two conditions account for 90% of cases of hypercalcaemia:

- 1. Primary hyperparathyroidism: commonest cause in non-hospitalised patients
- 2. Malignancy: the commonest cause in hospitalised patients. This may be due to a number of processes, including;
    - PTHrP from the tumour e.g. squamous cell lung cancer
    - bone metastases
    - myeloma,: due primarily to increased osteoclastic bone resorption caused by local cytokines (e.g. IL-1, tumour necrosis factor) released by the myeloma cells
- for this reason, measuring parathyroid hormone levels is the key investigation for patients with hypercalcaemia

Other causes include

- sarcoidosis
    - other causes of granulomas may lead to hypercalcaemia e.g. tuberculosis and histoplasmosis
- vitamin D intoxication
- acromegaly
- thyrotoxicosis
- Milk-alkali syndrome
- drugs:

    - thiazides
    - calcium-containing antacids
- dehydration
- Addison's disease
- Paget's disease of the bone
    - usually normal in this condition but hypercalcaemia may occur with prolonged immobilisation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 1 👎 1

Hypercalcaemia â€“ presentation and management

Suggest link

Report broken link

Media

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 4 👎 0

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

Report broken media

Score: **49.2%**

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A 55-year-old woman attends the clinic with refractory hypertension. A systematic enquiry was otherwise unremarkable. Her 7-day ambulatory mean blood pressure is 182/94 mmHg. She takes ramipril, amlodipine, and indapamide.

Blood results are as follows:

Na^+	142 mmol/L	(135 - 145)
K^+	2.5 mmol/L	(3.5 - 5.0)
Bicarbonate	36 mmol/L	(22 - 29)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)

Renin	3.2 mU/	(5.4 - 30)
Aldosterone	820 pmol/L	(100 - 450)

A CT scan is performed:

CT abdomen with contrast	Bilateral nodular adrenal hyperplasia

What treatment is indicated?

○ Amiloride ✕

○ Laparoscopic adrenalectomy ✕

○ Metyrapone ✕

○ Pituitary adenectomy ✕

○ Spironolactone ✕

Submit answer

Reference ranges ⌄

Score: **49.2%**

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What treatment is indicated?

- ○ Amiloride ×
- ○ Laparoscopic adrenalectomy ×
- ○ Metyrapone ×
- ○ Pituitary adenectomy ×
- ○ Spironolactone ×

Submit answer

Reference ranges ⌵

Score: **49.2%**

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Renin	3.2 mU/	(5.4 - 30)
Aldosterone	820 pmol/L	(100 - 450)

A CT scan is performed:

CT abdomen with contrast	Bilateral nodular adrenal hyperplasia

What treatment is indicated?

🟥	Amiloride	8%
	Laparoscopic adrenalectomy	8%
	Metyrapone	3%
	Pituitary adenectomy	1%
🟩	Spironolactone	80%

Primary hyperaldosteronism: manage with spironolactone

Important for me Less important

Spironolactone is correct. The patient has primary hyperaldosteronism secondary to bilateral adrenal hyperplasia. The first-line treatment for this condition is spironolactone (an aldosterone antagonist).

Amiloride is incorrect. Although this is a potassium-sparing diuretic like spironolactone, it has a very different mechanism of action. Its primary site of action is the distal tubule of the nephron where it selectively blocks sodium transport, thereby inhibiting sodium-potassium exchange. The mechanism of action of amiloride is independent of aldosterone.

Laparoscopic adrenalectomy is incorrect. Indications for this would be Conn's Syndrome (primary hyperaldosteronism secondary to a benign adrenal adenoma). The CT in this case has confirmed bilateral adrenal hyperplasia rather than an excisable adenoma.

Metyrapone is incorrect. Metyrapone is a competitive inhibitor of 11β-hydroxylation in the adrenal cortex; the resulting inhibition of cortisol (and to a lesser extent aldosterone) production leads to an increase in ACTH production which, in turn, leads to increased synthesis and release of cortisol precursors. Metyrapone can be used to treat resistant oedema due to increased aldosterone secretion in cirrhosis, nephrotic syndrome, and congestive heart failure, however, there is insufficient evidence to recommend its use in the management of primary hyperaldosteronism.

Pituitary adenectomy is incorrect. Cushing's disease occurs due to an ACTH-secreting pituitary adenoma resulting in high levels of cortisol and aldosterone. Although this remains within the differential diagnosis, there are no other clinical features (e.g. proximal myopathy, abdominal striae, weight gain) to support a diagnosis of Cushing's.

Discuss (1) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

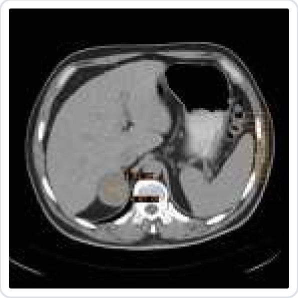
Causes
- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

Features
- **hypertension**
  - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
  - e.g. muscle weakness
  - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations
- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
  - hypertension with hypokalemia
  - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
  - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)

- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
  - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management
- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

Suggest link

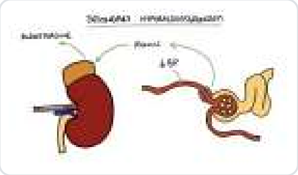
Report broken link

Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

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Question 61 of 217

A 21-year-old female presents with a vague two month history of lethargy, muscle pain and weight loss. Examination is unremarkable. Observations included heart rate 72/min, respiratory rate 14/min, oxygen saturations are 99% on air, blood pressure of 110/80mmHg. She was apyrexial.

Routine bloods are sent.

Hb	150 g/l	Na^+	128 mmol/l
Platelets	$200 * 10^9/l$	K^+	2.9 mmol/l
WBC	$12.0 * 10^9/l$	Urea	7.0 mmol/l
Neuts	$8.0 * 10^9/l$	Creatinine	85 µmol/l
Lymphs	$4.0 * 10^9/l$	CRP	11 mg/l

What is the next most appropriate investigation?

● Computed tomography (CT) of the thorax ×

○ Ultrasound of the renal tract ×

○ Serum aldosterone ×

○ Urinary electrolytes ×

○ Short synacthen test ×

Submit answer

Reference ranges

Score: **48.3%**

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Question 61 of 217 ✓

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Neuts	$8.0 * 10^9/l$	Creatinine	85 µmol/l
Lymphs	$4.0 * 10^9/l$	CRP	11 mg/l

What is the next most appropriate investigation?

Computed tomography (CT) of the thorax	4%
Ultrasound of the renal tract	3%
Serum aldosterone	13%
[✓ Urinary electrolytes]	52%
Short synacthen test	27%

The combination of lethargy, weight loss, low sodium and low potassium in a young patient suggest an inherited renal pathology such as Bartter's or Gitelman's syndrome. Liddle's syndrome is usually associated with high blood pressure.

Urinary electrolytes are a quick and simple test and would be likely to aid the diagnosis here.

Most other causes alluded to here would not cause both sodium and potassium to be low.

👍 | 👎 | Discuss (9) | Improve

Next question

Hypokalaemia and hypertension ★

For exams it is useful to be able to classify the causes of hypokalaemia in to those associated with hypertension, and those which are not

Hypokalaemia with hypertension
- Cushing's syndrome
- Conn's syndrome (primary hyperaldosteronism)
- Liddle's syndrome
- 11-beta hydroxylase deficiency*

Carbenoxolone, an anti-ulcer drug, and liquorice excess can potentially cause hypokalaemia associated with hypertension

Hypokalaemia without hypertension
- diuretics
- GI loss (e.g. Diarrhoea, vomiting)
- renal tubular acidosis (type 1 and 2**)
- Bartter's syndrome
- Gitelman syndrome

*21-hydroxylase deficiency, which accounts for 90% of congenital adrenal hyperplasia cases, is not associated with hypertension

**type 4 renal tubular acidosis is associated with hyperkalaemia

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypokalemia - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 2 👎 0

Report broken media

Score: **49.2%**

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A 24-year-old woman presents to the emergency department with dyspnoea. She has a past medical history of type 1 diabetes. She is poorly compliant with insulin treatment. She does not smoke or drink alcohol and lives with her family.

Her observations are heart rate 90 beats per minute, respiratory rate 24/minute, blood pressure 120/77 mmHg and temperature 36.7ºC.

Arterial blood gas:

pH	7.11	(7.35-7.45)
pO2	14.1 kPa	(11-13)
pCO2	3.4 kPa	(4.5-6)
HCO3	10 mmol/L	(22-26)
Glucose	24 mmol/L	(< 7.8)
Lactate	2.6 mmol/L	(0-2)
Potassium	4.8 mmol/L	(3.5-5.5)
Sodium	136 mmol/L	(135-145)

Blood ketones	3.4 mmol/L	(<0.6)

She is treated with intravenous normal saline with appropriate potassium replacement and a fixed rate insulin infusion (FRII) with normalization of her academia, ketonemia and hyperglycaemia.

What electrolyte abnormality is she at risk of developing during the resolution of this condition?

○ Hypercalcaemia ×

○ Hyperkalaemia ×

○ Hypernatraemia ×

○ Hypermagnesemia ×

○ Hypophosphataemia ×

Submit answer

Reference ranges ⌄

Score: **49.2%**

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HCO3	10 mmol/L	(22-26)
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She is treated with intravenous normal saline with appropriate potassium replacement and a fixed rate insulin infusion (FRII) with normalization of her academia, ketonemia and hyperglycaemia.

What electrolyte abnormality is she at risk of developing during the resolution of this condition?

Hypercalcaemia	2%
Hyperkalaemia	14%

Hypernatraemia	18%
Hypermagnesemia	2%
Hypophosphataemia	63%

Recovering DKA are at risk of hypophosphataemia

Important for me Less important

Hypophosphataemia is the correct answer. This patient has diabetic ketoacidosis (elevated blood glucose level, acidaemia and ketonemia). A rise in insulin due to exogenous insulin administration causes an intracellular shift of phosphate. There is an element of phosphaturia in addition.

Hypercalcaemia is incorrect. This has been noted to occur in the development of DKA, likely secondary to severe metabolic acidosis and insulin deficiency but would therefore be not expected to occur as DKA resolves.

Hyperkalaemia is incorrect. Most patients with DKA are potassium deficient and the risk is of hypokalaemia, not hyperkalaemia. Furthermore drives potassium into cells, resulting in hypokalaemia.

Hypermagnesemia is incorrect. Insulin administration causes uptake of magnesium into cells and may cause hypomagnesemia rather than hypermagnesemia.

Hyperphosphataemia is incorrect. It is low rather than high phosphate that is the risk in recovering DKA patients.

👍 👎 Discuss (7) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	**Volume**
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours

Fluid	Volume
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

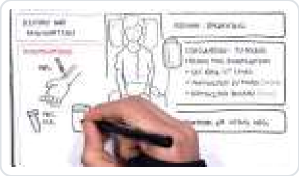
Links

Joint British Inpatient Diabetes Societies Inpatient Care Group 👍 33 👎 23

The Management of Diabetic ketoacidosis in adults

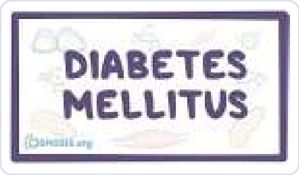
Suggest link Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **48.4%**

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A 28-year-old man presents to the GP with a 6-month history of excessive thirst and passing large volumes of dilute urine, including waking several times at night to urinate. He has no significant past medical history, his BMI is within the healthy range, and he denies taking any regular medications or recreational drugs. On examination, his vital signs are unremarkable and there are no clinical features of dehydration. Initial blood tests including glucose, calcium and renal function are normal. He is referred for further investigation.

The results of a water deprivation test are as follows:

Plasma osmolality (start)	300 mOsm/kg	(275 – 295)
Urine osmolality (after deprivation)	180 mOsm/kg	(>600)
Urine osmolality (post-desmopressin)	710 mOsm/kg	(>600)

Which of the following is the most likely diagnosis?

- ○ Cranial diabetes insipidus ×
- ○ Nephrogenic diabetes insipidus ×
- ○ Primary polydipsia ×
- ○ Type 2 diabetes mellitus ×
- ○ SIADH ×

Submit answer

Reference ranges

Score: **48.4%**

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Question 63 of 217 ✓

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Urine osmolality (post-desmopressin)	710 mOsm/kg	(>600)

Which of the following is the most likely diagnosis?

Cranial diabetes insipidus	85%
Nephrogenic diabetes insipidus	10%
Primary polydipsia	4%
Type 2 diabetes mellitus	0%
SIADH	1%

Water deprivation test: cranial DI

- urine osmolality after fluid deprivation: low
- urine osmolality after desmopressin: high

Important for me Less important

Cranial diabetes insipidus is the correct answer. This condition occurs due to deficiency of antidiuretic hormone (ADH), resulting in an inability to concentrate urine despite rising plasma osmolality. During water deprivation, urine remains dilute (<300 mOsm/kg), but after administration of desmopressin (a synthetic ADH analogue), there is a marked increase in urine osmolality (>600 mOsm/kg). This pattern—failure to concentrate urine during deprivation but robust response to desmopressin—is characteristic for cranial DI.

Nephrogenic diabetes insipidus would also present with polyuria and polydipsia; however, in this condition, there is renal resistance to ADH. Thus, both after fluid deprivation and after desmopressin administration, urine remains dilute (<300 mOsm/kg). The patient's strong response to desmopressin rules out nephrogenic DI.

Primary polydipsia involves excessive fluid intake leading to suppression of ADH secretion and secondary polyuria. In these patients, after water deprivation their urine osmolality will rise above 400 mOsm/kg as endogenous ADH starts concentrating the urine; post-desmopressin changes may be minimal or only slightly increased from baseline if maximal concentration already achieved. Here, the patient failed to concentrate urine until given desmopressin—a pattern not seen in primary polydipsia.

Type 2 diabetes mellitus can cause osmotic diuresis if glucose levels are very high but would be accompanied by hyperglycaemia on blood tests—which were reported as normal in this scenario—and does not fit with this pattern on a water deprivation test.

SIADH causes inappropriate retention of free water leading to concentrated urine even when it should be dilute; typically patients have low plasma sodium/osmolality with inappropriately high urine osmolality at baseline—not observed here—and would not respond dramatically to desmopressin.

👍 | 👎 | 💬 Discuss | Improve

Next question

Water deprivation test

The water deprivation test is designed to help evaluate patients who have polydipsia.

Method

- prevent patient drinking water
- ask the patient to empty their bladder
- hourly urine and plasma osmolalities

	Starting plasma osm.	Final urine osm.	Urine osm. post-DDAVP
Normal	Normal	> 600	> 600
Psychogenic polydipsia	Low	> 400	> 400
Cranial DI	High	< 300	> 600
Nephrogenic DI	High	< 300	< 300

+ Q 123

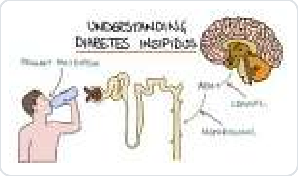
Next question

Textbooks

High-yield textbook

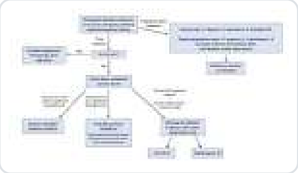
Extended textbook

Media


UNDERSTANDING DIABETES INSIPIDUS

Understanding Diabetes Insipidus

Zero to Finals - YouTube 👍 3 👎 0



An Approach to Polyuria

Strong Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **49.2%**

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← Question 64 of 217 →

A 42-year-old woman presents with feeling warm and more anxious than usual. She had an upper respiratory tract infection three weeks ago. On examination, you find a new tender goitre.

You send blood tests including thyroid function tests which are outlined in the table below.

Thyroid stimulating hormone (TSH)	0.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	25 pmol/L	(9.0 - 18)

Given the likely underlying diagnosis, what results do you expect from thyroid scintigraphy?

- ○ Globally normal uptake ×
- ○ Globally increased uptake ×
- ○ Globally reduced uptake ×
- ○ Well defined area of decreased uptake ×
- ○ Well defined area of increased uptake ×

Submit answer

Reference ranges

Score: **49.2%**

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Free thyroxine (T4)	25 pmol/L	(9.0 - 18)

Given the likely underlying diagnosis, what results do you expect from thyroid scintigraphy?

	Globally normal uptake	3%
	Globally increased uptake	16%
🟩	Globally reduced uptake	75%
🟥	Well defined area of decreased uptake	2%
	Well defined area of increased uptake	4%

De Quervain's thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

Important for me Less important

Subacute thyroiditis is thought to occur following viral infection and typically presents with hyperthyroidism and a new painful goitre. The investigation of choice is thyroid scintigraphy which reveals globally reduced uptake of iodine-131. Most cases are self-limiting and do not require treatment. The other answers provided are not the most common expected findings for De Quervain's thyroiditis.

Global normal uptake with uniform uptake of iodine-131 is most associated with a normal thyroid gland. Given the abnormal thyroid function and painful goitre, this would be an unexpected result in this case.

Globally increased uptake is most commonly associated with Grave's disease.

A well-defined area of decreased uptake would be most consistent with a cold (hypofunctioning) thyroid nodule. This finding would also not be consistent with the painful goitre or hyperthyroidism noted on blood tests in this case.

A well-defined area of increased uptake would be most consistent with a hot (hyperfunctioning) thyroid nodule.

👍 👎 Discuss (3) Improve

Next question

Subacute (De Quervain's) thyroiditis ★

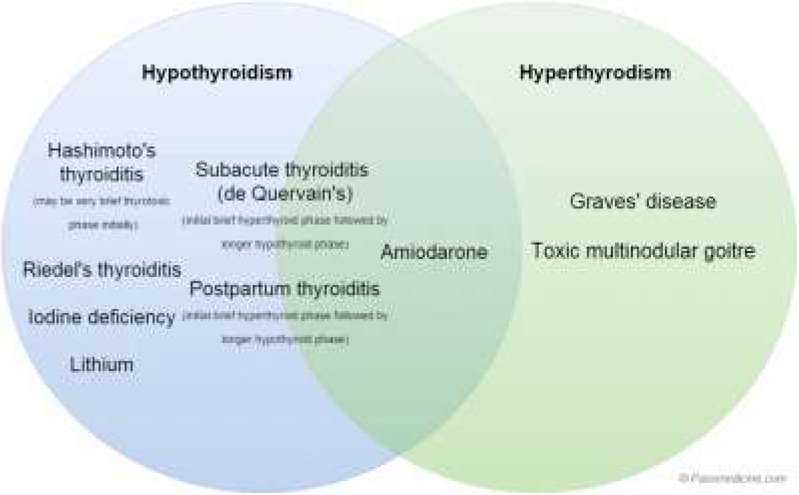
Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations
- thyroid scintigraphy: globally reduced uptake of iodine-131

Management

- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **48.4%**

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64 ✗

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Back to top

A 30-year-old female, who was diagnosed two months earlier with Graves disease and was started on carbimazole 40 mg per day , presented complaining of sore throat.

Investigations reveal:

Haemoglobin	11.5 g/dl
MCV	80 fl
White cell count	4.2 x 10^9/l
Neutrophils	2.0 x 10^9/l
Lymphocytes	2.3 x 10^9/l
Basophils	0.08 x 10^9/l
Eosinophils	0.1 x 10^9/l
Platelets	170 x 10^9/l

What is the most appropriate treatment for this patient?

- ○ Discontinue carbimazole and give propylthiouracil ✕
- ○ Discontinue carbimazole and give radioactive iodine ✕
- ○ Discontinue carbimazole and give antibiotics ✕
- ○ Reduce the dose of carbimazole ✕
- ○ Continue carbimazole ✕

Submit answer

Reference ranges

Score: **48.4%**

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Question 65 of 217 ✓

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Investigations reveal:

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Basophils	0.08 x 10^9/l
Eosinophils	0.1 x 10^9/l
Platelets	170 x 10^9/l

What is the most appropriate treatment for this patient?

Discontinue carbimazole and give propylthiouracil	29%
Discontinue carbimazole and give radioactive iodine	3%
Discontinue carbimazole and give antibiotics	18%
Reduce the dose of carbimazole	12%
Continue carbimazole	37%

Patients taking carbimazole may develop neutropaenia (although the incidence is very low) so they are warned about a sore throat while taking the medication.

This patient develpped a sore throat but her total white cell count and differential are normal, so she should be reassured and continue carbimazole.

If there is neutropaenia, carbimazole should be discontinued and substituted by propylthiouracil till the neutrophil count has recovered and then the patient is planned for radioactive iodine.

Discuss (10) | Improve

Next question

Carbimazole ★

Carbimazole is used in the management of thyrotoxicosis. It is typically given in high doses for 6 weeks until the patient becomes euthyroid before being reduced.

Mechanism of action

- blocks thyroid peroxidase from coupling and iodinating the tyrosine residues on thyroglobulin → reducing thyroid hormone production
- in contrast propylthiouracil as well as this central mechanism of action also has a peripheral action by inhibiting 5'-deiodinase which reduces peripheral conversion of T4 to T3

Adverse effects

- agranulocytosis
- crosses the placenta, but may be used in low doses during pregnancy

+ Q 123

Next question

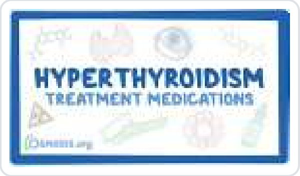
B I A

Textbooks

High-yield textbook

Extended textbook

Media


HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 6 👎 1

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Score: **49.2%**

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An 18-year-old boy attends your clinic with his parents. They have recently relocated from Bangladesh. His parents were advised to seek help after teachers at his school raised concerns about possible learning difficulties.

On examination, he is 1.42m tall and weighs 78kg. His BMI is 38.6kg/m². The cardio-respiratory examination is unremarkable, but you notice some shortening of the 5th metacarpals bilaterally when looking at his hands.

Blood tests have been completed and show:

Parathyroid hormone	78 pg/mL	(14-65)
Adjusted calcium	2.0 mmol/L	(2.2-2.6)
Phosphate	2.03 mmol/L	(0.97-1.45)

Given the above information, what is the most likely unifying diagnosis?

- ○ Hypoparathyroidism ✕
- ○ Osteogenesis imperfecta ✕
- ○ Osteomalacia ✕
- ○ Pseudohypoparathyroidism ✕
- ○ Pseudopseudohypoparathyroidism ✕

Submit answer

Reference ranges

Score: **49.2%**

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Question 66 of 217 ✓

An 18-year-old boy attends your clinic with his parents. They have recently relocated from Bangladesh. His parents were advised to seek help after teachers at his school raised concerns about possible learning difficulties.

On examination, he is 1.42m tall and weighs 78kg. His BMI is 38.6kg/m². The cardio-respiratory examination is unremarkable, but you notice some shortening of the 5th metacarpals bilaterally when looking at his hands.

Blood tests have been completed and show:

Parathyroid hormone	78 pg/mL	(14-65)
Adjusted calcium	2.0 mmol/L	(2.2-2.6)
Phosphate	2.03 mmol/L	(0.97-1.45)

Given the above information, what is the most likely unifying diagnosis?

Hypoparathyroidism	2%
Osteogenesis imperfecta	8%
Osteomalacia	5%
Pseudohypoparathyroidism	72%
Pseudopseudohypoparathyroidism	12%

Short fifth metacarpals, short stature, learning difficulties + ↑ PTH, ↓ calcium, ↑ phosphate → pseudohypoparathyroidism

Important for me Less important

The patient has short stature, short fifth metacarpals, and learning difficulties. With any presentation involving bony abnormalities, a bone profile is a helpful investigation. In this case, we see a raised parathyroid hormone, low calcium, and raised phosphate. The most likely diagnosis is, therefore, pseudohypoparathyroidism.

Pseudohypoparathyroidism is correct. The patient displays the phenotypical features of pseudohypoparathyroidism (short stature, short fifth metacarpals) in addition to learning difficulties. The blood tests also support this diagnosis. In pseudohypoparathyroidism, there is resistance to PTH. Low calcium levels promote PTH secretion, and the body responds by decreasing urinary calcium loss and releasing calcium from the bones. In pseudohypoparathyroidism, the body is not able to respond to increase calcium levels due to resistance to the PTH. We, therefore, see raised PTH (in response to low calcium), low calcium (as the body is resistant to the usual mechanism of increasing calcium levels), and raised phosphate (as PTH usually would increase phosphate excretion).

Pseudopseudohypoparathyroidism is incorrect. In pseudopseudohypoparathyroidism, you often see skeletal abnormalities and phenotypical features of type 1A pseudohypoparathyroidism, however, the biochemistry is normal. This is due to the genetic phenomenon of imprinting. Given the patient in the stem has abnormal biochemistry, the diagnosis is not pseudopseudohypoparathyroidism.

Osteomalacia is incorrect. This is a deficiency in vitamin D. Biochemical abnormalities include high PTH, and low phosphate and calcium. This is not in keeping with the biochemical abnormalities seen in this patient, and would not explain the learning difficulties and other physical abnormalities.

Osteogenesis imperfecta is incorrect. This is a genetic condition of type I collagen that predisposes to reduced bone strength. It is also termed 'brittle bone disease'. You would not see abnormalities in calcium, phosphate, or PTH levels, and so this is not the correct answer here.

Hypoparathyroidism is incorrect. In hypoparathyroidism, you would see low PTH levels and this can be caused by a number of factors e.g. autoimmune disease, inadvertent removal of the parathyroid glands during thyroidectomy, or haemochromatosis amongst others. This patient has raised PTH and so this is not the correct answer. Hypoparathyroidism would also be associated with raised calcium and low phosphate levels.

👍 | 👎 | Discuss (4) | Improve

Next question

Pseudohypoparathyroidism ★

Pseudohypoparathyroidism is caused by target cell insensitivity to parathyroid hormone (PTH) due to a mutation in a G-protein. In type I pseudohypoparathyroidism there is a complete receptor defect whereas in type II the cell receptor is intact. Pseudohypoparathyroidism is typically inherited in an autosomal dominant fashion.

Bloods
- ↑ PTH
- ↓ calcium
- ↑ phosphate

Features
- short fourth and fifth metacarpals
- short stature
- learning difficulties
- obesity
- round face

Investigation
- an infusion of PTH followed by measurement of urinary phosphate and cAMP measurement - this can help differentiate between type I (neither phosphate or cAMP levels rise) and II (cAMP rises but phosphate levels do not change)

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **50%**

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A 28-year-old woman presents to her GP with a 3-month history of fatigue, muscle cramps, and occasional palpitations. She has no significant past medical history and takes no regular medications. Her blood pressure is 115/75 mmHg. Initial blood tests reveal:

Na+	138 mmol/L	(135-145)
K+	2.8 mmol/L	(3.5-5.0)
Cl-	95 mmol/L	(95-105)
HCO3-	32 mmol/L	(22-29)
Mg2+	0.55 mmol/L	(0.7-1.0)
Ca2+	2.35 mmol/L	(2.2-2.6)
Creatinine	75 μmol/L	(45-90)

Urinary calcium is low at 1.2 mmol/24h (normal range 2.5-7.5 mmol/24h). Further investigations are being considered to establish a diagnosis.

What is the most appropriate next diagnostic test?

- ○ 24-hour urinary potassium measurement ×
- ○ Plasma renin and aldosterone levels ×
- ○ Genetic testing for mutations in the SLC12A3 gene ×
- ○ Renal ultrasound scan ×
- ○ Diuretic challenge test with thiazides ×

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **50%**

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67 -

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A 28-year-old woman presents to her GP with a 3-month history of fatigue, muscle cramps, and occasional palpitations. She has no significant past medical history and takes no regular medications. Her blood pressure is 115/75 mmHg. Initial blood tests reveal:

Na+	138 mmol/L	(135-145)
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Mg2+	0.55 mmol/L	(0.7-1.0)
Ca2+	2.35 mmol/L	(2.2-2.6)
Creatinine	75 µmol/L	(45-90)

Urinary calcium is low at 1.2 mmol/24h (normal range 2.5-7.5 mmol/24h). Further investigations are being considered to establish a diagnosis.

What is the most appropriate next diagnostic test?

24-hour urinary potassium measurement	5%
Plasma renin and aldosterone levels	14%
Genetic testing for mutations in the SLC12A3 gene	74%
Renal ultrasound scan	1%
Diuretic challenge test with thiazides	5%

Diagnostic test for Gitelman's syndrome: genetic testing for mutations in the SLC12A3 gene

Important for me Less important

Genetic testing for mutations in the SLC12A3 gene is the most appropriate next diagnostic test in this case. The patient presents with a clinical picture highly suggestive of Gitelman's syndrome: hypokalaemia, metabolic alkalosis (elevated bicarbonate), hypomagnesaemia, and hypocalciuria. Gitelman's syndrome is an autosomal recessive tubulopathy caused by mutations in the SLC12A3 gene, which encodes the thiazide-sensitive sodium-chloride cotransporter in the distal convoluted tubule. This leads to renal salt wasting similar to the effect of thiazide diuretics. The patient's normal blood pressure, despite electrolyte abnormalities, is also characteristic of Gitelman's syndrome (as opposed to primary hyperaldosteronism). Genetic testing is considered the gold standard for diagnosis, with approximately 80-90% of patients having identifiable mutations in the SLC12A3 gene. Confirming the diagnosis is important for appropriate management and genetic counselling.

24-hour urinary potassium measurement would show increased potassium excretion, which would be consistent with Gitelman's syndrome but is not specific enough to establish the diagnosis. While this test might help confirm inappropriate renal potassium wasting, it would not differentiate between various causes of hypokalaemia such as Gitelman's syndrome, Bartter syndrome, or diuretic abuse. The clinical picture already strongly suggests renal potassium wasting, so this test would add little additional diagnostic value at this stage.

Plasma renin and aldosterone levels would likely show elevated renin and secondary hyperaldosteronism in Gitelman's syndrome due to volume depletion. However, these findings are not specific to Gitelman's syndrome and can be seen in other conditions causing volume depletion or renal salt wasting. These tests are more useful in excluding primary hyperaldosteronism, which typically presents with hypertension rather than the normotension seen in this patient.

Renal ultrasound scan would be normal in Gitelman's syndrome as it is a functional rather than structural renal disorder. This investigation would not contribute to establishing the diagnosis in this case. Renal ultrasound might be useful to exclude other causes of electrolyte disturbances such as obstructive uropathy or renal artery stenosis, but these conditions would typically present with different clinical and biochemical features.

Diuretic challenge test with thiazides is not appropriate in this context. Patients with Gitelman's syndrome have a genetic defect in the thiazide-sensitive sodium-chloride cotransporter, essentially mimicking the effect of chronic thiazide diuretic use. Therefore, they would show a blunted natriuretic response to thiazide diuretics. While this test could theoretically help distinguish Gitelman's syndrome from diuretic abuse, it is invasive, potentially dangerous in an already hypokalaemic patient, and has been largely replaced by genetic testing in clinical practice.

👍 👎 💬 Discuss Improve

Next question

Gitelman's syndrome ★

Gitelman's syndrome is due to a defect in the thiazide-sensitive Na$^+$ Cl- transporter in the distal convoluted tubule.

Features
- normotension
- hypokalaemia
- hypocalciuria
- hypomagnesaemia
- metabolic alkalosis

Diagnosis
- genetic testing for mutations in the SLC12A3 gene

Management
- oral potassium and magnesium supplementation
- potassium-sparing diuretics may be used if still hypokalaemic/symptomatic

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **49.3%**

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Question 68 of 217

A 45-year-old man presents to the endocrine clinic for review. He has had 3 stones in weight gain over the past 6 months and his GP is concerned about a possible diagnosis of Cushing's syndrome and has checked an initial 24hr urinary free cortisol which is elevated. He is hypertensive with a blood pressure of 155/90 mmHg, his pulse is 75 beats per minute and regular. His body mass index is 35 kg/m² and there are obvious abdominal striae. Which of the following would be most suggestive of an adrenal adenoma producing cortisol?

- ○ Normal 9am serum cortisol ×
- ○ Raised urinary free cortisol on repeat testing ×
- ○ Serum cortisol of 220 mmol/l at 9am after an overnight dexamethasone suppression test ×
- ○ Serum potassium of 2.4 mmol/l ×
- ○ Undetectable levels of ACTH ×

Submit answer

Reference ranges

Score: **49.3%**

1 ×
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3 ×

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A 45-year-old man presents to the endocrine clinic for review. He has had 3 stones in weight gain over the past 6 months and his GP is concerned about a possible diagnosis of Cushing's syndrome and has checked an initial 24hr urinary free cortisol which is elevated. He is hypertensive with a blood pressure of 155/90 mmHg, his pulse is 75 beats per minute and regular. His body mass index is 35 kg/m² and there are obvious abdominal striae. Which of the following would be most suggestive of an adrenal adenoma producing cortisol?

Normal 9am serum cortisol	1%
Raised urinary free cortisol on repeat testing	5%
Serum cortisol of 220 mmol/l at 9am after an overnight dexamethasone suppression test	32%
Serum potassium of 2.4 mmol/l	3%
Undetectable levels of ACTH	60%

The key fact is that an adrenal adenoma producing cortisol would suppress the release of ACTH. For this reason, if ACTH is undetectable then an adrenal tumour is the most likely cause. In this case, a CT abdomen would be the obvious next step.

Normal 9 am serum cortisol would make a diagnosis of Cushing's less likely, and a raised urinary free cortisol on repeat testing is not specific for the underlying cause. Failure to suppress after overnight dexamethasone suppression test merely confirms a diagnosis of Cushing's. Serum potassium levels as low as 2.4 mmol/l would be very unusual in a case of Cushing's.

Discuss (5) | Improve

Cushing's syndrome: investigations ★

This section will consider:

- the general lab findings consistent with Cushing's syndrome
- tests to confirm whether a patient indeed has Cushing's syndrome
- tests to find the underlying cause of Cushing's syndrome

It is useful to bear in mind the possible causes:

- iatrogenic: corticosteroid therapy
- ACTH-dependent causes
  - Cushing's disease (a pituitary adenoma → ACTH secretion)
  - ectopic ACTH secretion secondary to a malignancy
- ACTH-independent causes
  - adrenal adenoma

General findings consistent with Cushing's syndrome

A hypokalaemic metabolic alkalosis may be seen, along with impaired glucose tolerance.

Ectopic ACTH secretion (e.g. secondary to small cell lung cancer) is characteristically associated with very low potassium levels.

Tests to confirm Cushing's syndrome

The three most commonly used tests are:

- overnight (low-dose) dexamethasone suppression test
  - this is the most sensitive test and is now used first-line to test for Cushing's syndrome
  - patients with Cushing's syndrome do not have their morning cortisol spike suppressed
- 24 hr urinary free cortisol
  - two measurements are required
- bedtime salivary cortisol

    - two measurements are required

Localisation tests

The first-line localisation is 9am and midnight plasma ACTH (and cortisol) levels. If ACTH is suppressed then a non-ACTH dependent cause is likely such as an adrenal adenoma

High-dose dexamethasone suppression test

The high-dose dexamethasone suppression test may be used to localise the pathology resulting in Cushing's syndrome. This test may be interpreted as follows:

Cortisol	ACTH	Interpretation
Not suppressed	Suppressed	Cushing's syndrome due to other causes (e.g. adrenal adenomas)
Suppressed	Suppressed	Cushing's disease (i.e. pituitary adenoma → ACTH secretion)
Not suppressed	Not suppressed	Ectopic ACTH syndrome

Other tests

CRH stimulation
- if pituitary source then cortisol rises
- if ectopic/adrenal then no change in cortisol

Petrosal sinus sampling of ACTH may be needed to differentiate between pituitary and ectopic ACTH secretion.

An insulin stress test is used to differentiate between true Cushing's and pseudo-Cushing's.

+ Q 123

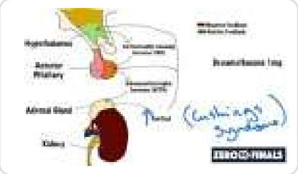
Next question

Textbooks

High-yield textbook

Extended textbook

Media


Hypothalamus
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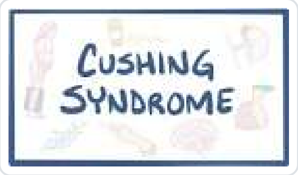
Tumo (Cushing's Syndrome)
ZERO TO FINALS

Understanding The Dexamethasone Suppression Test

Zero to Finals - YouTube 👍 31 👎 3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Cushing syndrome

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CUSHING'S
SYNDROME

Understanding Cushing's Syndrome

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Report broken media

Score: **48.5%**

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67	✗
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A 48-year-old woman presents with a 2-month history of fatigue, anorexia and nausea. She has also noticed some darkening of the skin on her hands. Prior to this, she was fit and well, with no significant past medical history. She has a strong family history of Hashimoto's thyroiditis.

On examination, there is some evidence of wasting of the face, and noticeable hyperpigmentation of the palmar creases. Her blood pressure is 105/75 mmHg. Blood tests are taken:

Na^+	134 mmol/L	(135 - 145)
K^+	5.3 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	76 µmol/L	(55 - 120)
Calcium	2.43 mmol/L	(2.1-2.6)
Thyroid stimulating hormone (TSH)	1.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	13.3 pmol/L	(9.0 - 18)

An initial test is conducted to confirm the likely diagnosis.

Which subsequent test can be performed to distinguish a primary cause from a secondary cause?

- ○ High-dose dexamethasone suppression test ×
- ○ Long Synacthen test ×
- ○ Low-dose dexamethasone suppression test ×
- ○ Short Synacthen test ×

○ Urinary metanephrines ✕

Submit answer

Reference ranges ⌄

Score: **48.5%**

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A 48-year-old woman presents with a 2-month history of fatigue, anorexia and nausea. She has also noticed some darkening of the skin on her hands. Prior to this, she was fit and well, with no significant past medical history. She has a strong family history of Hashimoto's thyroiditis.

On examination, there is some evidence of wasting of the face, and noticeable hyperpigmentation of the palmar creases. Her blood pressure is 105/75 mmHg. Blood tests are taken:

Na^+	134 mmol/L	(135 - 145)
K^+	5.3 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	76 µmol/L	(55 - 120)
Calcium	2.43 mmol/L	(2.1-2.6)
Thyroid stimulating hormone (TSH)	1.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	13.3 pmol/L	(9.0 - 18)

An initial test is conducted to confirm the likely diagnosis.

Which subsequent test can be performed to distinguish a primary cause from a secondary cause?

	High-dose dexamethasone suppression test	17%
✓	Long Synacthen test	44%
	Low-dose dexamethasone suppression test	5%
	Short Synacthen test	33%

Urinary metanephrines 0%

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

Important for me Less important

The diagnosis here is that of adrenal insufficiency. The history of fatigue, anorexia and nausea points towards this, along with evidence of hyperpigmentation of the palmar creases, hypotension, hyponatraemia and hyperkalaemia. A primary failure is known as Addison's disease. To make the initial diagnosis of adrenal failure, a short Synacthen test should be used - cortisol will fail to rise. However, the question is asking specifically how to differentiate primary from secondary adrenal failure, secondary being a failure of the pituitary to make sufficient ACTH. For this, the long Synacthen test should be used - a higher dose is given and then cortisol levels measured over a longer time period. With primary failure, the adrenals will still be unable to produce cortisol, but with secondary failure, the exogenous ACTH will eventually push the adrenals to produce cortisol.

The high-dose dexamethasone suppression test is used to differentiate primary and secondary causes of Cushing's syndrome, not adrenal insufficiency. This would have instead presented with weight gain and hypokalemia, not the other way around.

The low-dose dexamethasone suppression test is used first-line to make the initial diagnosis of Cushing's. It does not play a role here.

The short Synacthen test is used to initially diagnose adrenal insufficiency. It does not, however, differentiate between primary and secondary causes - the long test does this.

Urinary metanephrines are used to diagnose pheochromocytoma. In this condition, there is increased secretion of catecholamines and patients present with hypertension, headaches, sweating and palpitations.

👍 👎 Discuss (6) Improve

Next question

Addison's disease: investigations

In a patient with suspected Addison's disease the definite investigation is an ACTH stimulation test (short Synacthen test). Plasma cortisol is measured before and 30 minutes after giving Synacthen 250ug IM. Adrenal autoantibodies such as anti-21-hydroxylase may also be demonstrated.

If an ACTH stimulation test is not readily available (e.g. in primary care) then sending a 9 am serum cortisol can be useful:

- > 500 nmol/l makes Addison's very unlikely
- < 100 nmol/l is definitely abnormal
- 100-500 nmol/l should prompt a ACTH stimulation test to be performed

Associated electrolyte abnormalities are seen in around one-third of undiagnosed patients:

- hyperkalaemia
- hyponatraemia
- hypoglycaemia
- metabolic acidosis

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 14 👎 7

2017 Adrenal insufficiency â€“ recognition and management

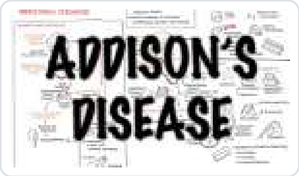
Suggest link

Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 2 👎 1



Addison's disease

Armando Hasudungan - YouTube 👍 2 👎 1

Report broken media

Score: **49.3%**

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A 28-year-old lady has noticed over the last year that she does not tolerate cold weather well. She is fatigued and her partner notices that she is also low in mood despite having no obvious triggers. Physical examination is unremarkable. Her electrocardiogram (ECG) demonstrates a sinus rhythm at 43 beats per minute. She has a background of type 1 diabetes mellitus for which she takes insulin. She also has coeliac disease. Her blood results are shown below:

Hb	136 g/l
MCV	103 fL

Na	133 mmol/l
K	4 mmol/l
Urea	3.5 mmol/l
Creatinine	70 µmol/l

Glycosylated haemoglobin (HbA1c)	51 mmol/mol (6.8%)
TSH	9.2 mIU/L (reference range 0.3-4.0 mIU/L
T3	2 pmol/L (reference range 3-9 pmol/L)
T4	5 pmol/L (reference range 9-25 pmol/L)

What is the next best step in her management?

○ Commence levothyroxine

○ Commence carbimazole

○ Ultrasound scan thyroid

○ MRI pituitary

○ Short synacthen test ✕

Submit answer

Reference ranges ⌄

Score: **49.3%**

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T3	2 pmol/L (reference range 3-9 pmol/L)
T4	5 pmol/L (reference range 9-25 pmol/L)

What is the next best step in her management?

Commence levothyroxine	63%
Commence carbimazole	1%
Ultrasound scan thyroid	7%
MRI pituitary	4%

Short synacthen test 24%

The short synacthen test is the best test to diagnose Addison's disease

Important for me Less important

Every time you see type 1 diabetes, pernicious anaemia, rheumatoid arthritis, coeliac, or indeed ANY of the autoimmune conditions in the past medical history of a patient in the exam engage autoimmune mode in your brain. Once you have done that, you will be specifically looking not to miss other coexistent autoimmune conditions they may be trying to hide from you behind a cryptic clinical sign or some subtle blood abnormality. In this example, where she has type 1 diabetes, thinking like that then gives you the diagnosis of an autoimmune hypothyroid cause (Hashimoto's). If you think you have made a diagnosis of Hashimoto's, you should routinely next always look to rule out Addison's, even if the sodium is normal (which it isn't in this example: a subtle blood abnormality). Addison's (just like other autoimmune conditions) may coexist with Hashimoto's. However, the danger of missing Addison's in Hashimoto's is that it sits there masked by the hypothyroid. If you miss it and treat the hypothyroid blindly first without covering the Addison's, you will unmask the Addison's and the patient will come back in an adrenal crisis a few days later.

👍 👎 💬 Discuss (16) Improve

Next question

Addison's disease: investigations ★

In a patient with suspected Addison's disease the definite investigation is an ACTH stimulation test (short Synacthen test). Plasma cortisol is measured before and 30 minutes after giving Synacthen 250ug IM. Adrenal autoantibodies such as anti-21-hydroxylase may also be demonstrated.

If an ACTH stimulation test is not readily available (e.g. in primary care) then sending a 9 am serum cortisol can be useful:

- > 500 nmol/l makes Addison's very unlikely
- < 100 nmol/l is definitely abnormal
- 100-500 nmol/l should prompt a ACTH stimulation test to be performed

Associated electrolyte abnormalities are seen in around one-third of undiagnosed patients:

- hyperkalaemia
- hyponatraemia
- hypoglycaemia
- metabolic acidosis

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 14 👎 7

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 2 👎 1

Addison's disease

Armando Hasudungan - YouTube 👍 2 👎 1

Report broken media

Score: **48.6%**

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A 43-year-old woman with a background of anaemia and epilepsy is referred to the endocrinology clinic with fatigue, weight loss, abdominal pain, nausea, and vomiting. She does not take any steroids. On examination, there is no evidence of any abnormal pigmentation. A series of cortisol measurements are taken after 250µg of synacthen injection at 9 am:

Baseline cortisol	190 nmol/L	(>180 nmol/L)
Cortisol at 30min	390 nmol/L	(>420 nmol/L)
Cortisol at 60min	520 nmol/L	(>420 nmol/L)

What is the most appropriate next investigation?

- ○ Insulin tolerance test
- ○ ~~Long synacthen test~~
- ○ Oral glucose tolerance test
- ● Overnight dexamethasone suppression test
- ○ ~~Water deprivation test~~

Submit answer

Reference ranges

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Question 71 of 217 ✓

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What is the most appropriate next investigation?

Insulin tolerance test	11%
Long synacthen test	60%
Oral glucose tolerance test	8%
Overnight dexamethasone suppression test	20%
Water deprivation test	1%

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

Important for me Less important

This patient has signs of adrenal failure and undergoes a short synacthen test.

Generally, a cortisol of >420nmol/L at 30 or 60 minutes post-synacthen indicates an adequate adrenal response. The test in this patient shows a borderline response. Although this makes primary adrenal failure less likely, the short synacthen test is less sensitive to secondary adrenal failure. It is important to investigate further when results are borderline and there is a strong clinical history. Generally, secondary adrenal insufficiency presents similarly to Addison's disease but with less hypovolaemia and no pigmentation.

A long synacthen test (where samples are taken at 1, 4, 8, and 24 hours) can be used to distinguish Addison's from secondary causes of adrenal insufficiency such as steroid use, panhypopituitarism, and isolated failure of adrenocorticotrophic hormone (ACTH).

The insulin tolerance test to the point of extreme hypoglycemia is considered the gold standard for assessing the integrity of the hypothalamic-pituitary-adrenal axis. It can be used in the diagnosis of panhypopituitarism. However, this test is dangerous and contraindicated in those with epilepsy or ischaemic heart disease.

The oral glucose tolerance test determines how quickly glucose is cleared from the blood. It is usually used to test for diabetes, insulin resistance, or impaired beta-cell function. It can also be used alongside insulin-like growth factor 1 (IGF-1) levels to diagnose acromegaly.

The overnight dexamethasone suppression test is useful in identifying adrenal hyperfunction (Cushing's syndrome) or failure (Addison's). It is unlikely to be useful after a negative short synacthen test, which is the gold standard for diagnosis of Addison's.

The water deprivation test is used in the diagnosis of diabetes insipidus.

👍 👎 Discuss (6) Improve

Next question

Addison's disease: investigations ★

In a patient with suspected Addison's disease the definite investigation is an ACTH stimulation test (short Synacthen test). Plasma cortisol is measured before and 30 minutes after giving Synacthen 250ug IM. Adrenal autoantibodies such as anti-21-hydroxylase may also be demonstrated.

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AI + Q 123

Next question

Textbooks

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Links

Royal College of Physicians 👍 14 👎 7

2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 2 👎 1

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 2 👎 1

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Score: **49.3%**

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A 24-year-old woman attends her GP with fatigue and muscle cramps. She has no significant past medical history and is not on any regular medication. Her blood pressure is 108/68 mmHg. On examination, she appears well and her cardiovascular and respiratory examinations are unremarkable. Urinalysis shows low urinary calcium excretion. The following laboratory results are available:

Na^+	139 mmol/L	(135 - 145)
K^+	2.7 mmol/L	(3.5 - 5.0)
Mg^{2+}	0.53 mmol/L	(0.7 - 1.0)
HCO_3^-	29 mmol/L	(22 - 28)
Creatinine	67 μmol/L	(55 - 120)
Ca^{2+} (urine)	Low	

What is the most appropriate first-line management for this patient?

- ○ Oral potassium and magnesium supplementation ×
- ○ Thiazide diuretic ×
- ○ Intravenous potassium replacement ×
- ○ Fludrocortisone ×
- ○ Potassium-sparing diuretic ×

Submit answer

Reference ranges

Score: **49.3%**

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HCO_3^-	29 mmol/L	(22 - 28)
Creatinine	67 µmol/L	(55 - 120)
Ca^{2+} (urine)	Low	

What is the most appropriate first-line management for this patient?

🟩	Oral potassium and magnesium supplementation	61%
🟥	Thiazide diuretic	16%
	Intravenous potassium replacement	5%
	Fludrocortisone	2%
	Potassium-sparing diuretic	16%

Oral potassium and magnesium supplementation is the first line management of Gitelman's syndrome

Important for me Less important

Oral potassium and magnesium supplementation: This is the correct answer as it represents the first-line management of Gitelman's syndrome, which this patient likely has based on her clinical presentation—young age, normotension, hypokalaemia, hypomagnesaemia, metabolic alkalosis, and low urinary calcium excretion. Oral supplementation addresses both electrolyte abnormalities directly and is recommended by UK guidelines as initial therapy before considering other agents.

Thiazide diuretic: This would be inappropriate in Gitelman's syndrome because the condition itself results from a defect in the thiazide-sensitive sodium-chloride co-transporter; giving a thiazide would worsen electrolyte loss rather than improve it.

Intravenous potassium replacement: While IV potassium may be used in severe or symptomatic hypokalaemia, oral replacement is preferred for chronic management in stable patients such as this one seen in primary care with mild symptoms.

Fludrocortisone: This mineralocorticoid would promote further renal potassium wasting by increasing distal tubular sodium reabsorption at the expense of potassium excretion; thus it would exacerbate hypokalaemia rather than treat it.

Potassium-sparing diuretic: Although these agents can be considered if oral supplements are insufficient to control symptoms or biochemical abnormalities, they are not first-line therapy according to UK guidance; they are reserved for refractory cases after oral supplementation has been tried.

👍 👎 Discuss Improve

Next question

Gitelman's syndrome

Gitelman's syndrome is due to a defect in the thiazide-sensitive Na^+ Cl- transporter in the distal convoluted tubule.

Features

- normotension
- hypokalaemia
- hypocalciuria
- hypomagnesaemia
- metabolic alkalosis

Diagnosis

- genetic testing for mutations in the SLC12A3 gene

Management

- oral potassium and magnesium supplementation
- potassium-sparing diuretics may be used if still hypokalaemic/symptomatic

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **48.6%**

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A 19-year-old pharmacy student is admitted to hospital after collapsing while at work. She denies biting her tongue or becoming incontinent during the collapse and was groggy but alert on coming around. At the time, a first aider measured her blood glucose to be 1.5 mmol/l. The patients mother reports that the patient has had 2 other episodes of collapse.

The students observations include a blood pressure of 127/77 mmHg, pulse of 81 bpm, and oxygen sats of 97%.

What is the best first-line investigation?

- ○ Glucose, c-peptide and insulin ✕
- ○ Morning c-peptide ✕
- ○ Evening c-peptide ✕
- ○ Computed tomography (CT) scan of the abdomen ✕
- ○ Oral glucose tolerance test ✕

Submit answer

Reference ranges

Score: **48.6%**

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The students observations include a blood pressure of 127/77 mmHg, pulse of 81 bpm, and oxygen sats of 97%.

What is the best first-line investigation?

[✓ Glucose, c-peptide and insulin]	91%
Morning c-peptide	6%
Evening c-peptide	1%
Computed tomography (CT) scan of the abdomen	1%
Oral glucose tolerance test	1%

Measuring blood glucose, insulin and c-peptide allows the differentiation between the causes of hypoglycaemic attacks, including insulinoma, insulin or sulphonylurea misuse.

Discuss (7) | Improve

Next question

Hypoglycaemia

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
  - causes exaggerated insulin secretion
  - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia

- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later
- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic) neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features

- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
  - Sweating
  - Shaking
  - Hunger
  - Anxiety
  - Nausea
- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
  - Weakness
  - Vision changes

    - Confusion
    - Dizziness
- Severe and uncommon features of hypoglycaemia include:
    - Convulsion
    - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
    - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form

    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
    - If the patient is alert, a quick-acting carbohydrate may be given (as above)
    - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
    - Alternatively, intravenous 20% glucose solution may be given through a large vein

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **49.3%**

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73 ✓

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A 45-year-old female presents with a 2-year history of headache and visual blurring. When initially presenting to her GP 2 years ago her blood pressure was found to be 235/160mmHg. Subsequently, despite maximal doses of four anti-hypertensives, including 50mg spironolactone, her blood pressure remains poorly controlled. Her latest blood tests demonstrate the following:

Na^+	140 mmol/l
K^+	2.9 mmol/l
Urea	5.8 mmol/l
Creatinine	78 µmol/l
CRP	2 mg/l

Serum ambulatory renin activity	0.34 pmol/L @ 3-4 hours (normal range 0.8-3.5 pmol/ml/hr)
Serum ambulatory aldosterone	2052 pmol/L@ 3-4 hours (normal range 100-800)

A CT adrenal reveals a right adrenal mass of 2.5cm diameter. The patient is keen to take away the underlying problem. What is the most appropriate next management step?

- ○ Add amiloride ×
- ○ Add eplerenone ×
- ○ Increase spironolactone to 100mg OD ×
- ○ Adrenal vein sampling ×
- ● Right adrenalectomy ×

Submit answer

Reference ranges

Score: **49.3%**

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Serum ambulatory aldosterone	2052 pmol/L@ 3-4 hours (normal range 100-800)

A CT adrenal reveals a right adrenal mass of 2.5cm diameter. The patient is keen to take away the underlying problem. What is the most appropriate next management step?

Add amiloride	5%
Add eplerenone	3%
Increase spironolactone to 100mg OD	7%
Adrenal vein sampling	33%
Right adrenalectomy	53%

Adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism

Important for me Less important

The patient presents with poorly controlled hypertension, is hypokalaemic, hypertensive, metabolically alkalotic, has increased aldosterone and reduced renin. The underlying diagnosis is Conn's disease. The treatment involves removal of the aldosterone secreting tumour. However, those pursuing adrenalectomy must undergo adrenal vein sampling first, CT imaging alone is not diagnostic of the symptomatic side responsible for aldosterone secretion.

👍 👎 Discuss (18) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes

- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

Features

- **hypertension**

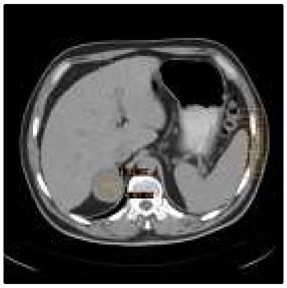
    - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
    - e.g. muscle weakness
    - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
    - hypertension with hypokalemia
    - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
    - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
    - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

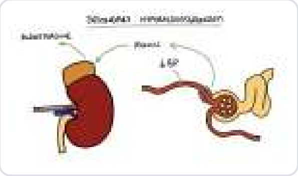
Suggest link Report broken link

Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



[Hyperaldosteronism and Conn's Syndrome]

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

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Question 75 of 217

A 41-year-old woman is admitted to hospital with acute epigastric abdominal pain that radiates to her back. She has nausea but has not vomited. A diagnosis of acute pancreatitis is suspected and she is commenced on intravenous fluids. Her observations include a blood pressure of 129/72 mmHg, pulse of 88 bpm, and oxygen sats of 97%.

Blood tests are performed and reveal:

Hb	13.9 g/l
Platelets	194 * 10^9/l
WBC	8.6 * 10^9/l
Na^+	139 mmol/l
K^+	4.2 mmol/l
Urea	4.1 mmol/l
Creatinine	92 µmol/l
Bilirubin	10 µmol/l
ALP	39 u/l
ALT	34 u/l
γGT	44 u/l
Albumin	48 g/l
Triglycerides	12.1 mmol/l
HDL cholesterol	1.1 mmol/l
LDL cholesterol	3.5 mmol/l

What is the most appropriate treatment for this patients condition?

○ Atorvastatin ×

○ Fenofibrate ×

○ Lovastatin ×

○ Ezetimibe ×

○ Alirocumab ×

Submit answer

Reference ranges ⌄

Score: **50%**

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PassMedicine

Question 75 of 217 ✓

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ALP	39 u/l
ALT	34 u/l
γGT	44 u/l
Albumin	48 g/l
Triglycerides	12.1 mmol/l
HDL cholesterol	1.1 mmol/l
LDL cholesterol	3.5 mmol/l

What is the most appropriate treatment for this patients condition?

Atorvastatin	15%
Fenofibrate	70%

Lovastatin	1%
Ezetimibe	12%
Alirocumab	3%

Fibrates are the most effective drug for treating hypertriglyceridaemia

Important for me Less important

The diagnosis is hypertriglyceridaemia, which has caused this patients acute pancreatitis. Fibrates are the treatment for hypertriglyceridaemia at high enough levels to cause acute pancreatitis.

👍 👎 Discuss (7) Improve

Next question

Primary hypertriglyceridaemia ★

Usually due to polygenic factors

May also be due to lipoprotein lipase deficiency

Management
- fibrates are generally used first-line
- statins do reduce triglyceride levels and they may be indicated, particularly if there is mixed hyperlipidaemia

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **50.7%**

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Question 76 of 217

You are called to see a 35-year-old man on the Acute Medical Unit who is having a seizure.

He was admitted 2 days ago having presented acutely agitated to the Emergency Department. He gave a 10-year history of alcohol overuse and at that time had not had an alcoholic drink in 24 hours following an argument with his girlfriend where he vowed to give up alcohol. Prior to this he had been drinking 4-6 litres of cider per day plus additional spirits in variable amounts. He was admitted for detox and prescribed chlordiazepoxide, pabrinex and fluids. He has no other past medical history.

Nursing staff inform you that the patient has had no other seizures on this admission and has been eating and drinking small amounts today. However, he has been complaining of all over body pain and became confused 2-3 hours ago. The seizure is generalised tonic-clonic and self terminates after 3 minutes.

On examination post-seizure the patient is drowsy but responding to voice. His saturations are 100% on 15 litres oxygen via non-rebreathe mask and his temperature is 37.2 ºC. His heart rate is 110 beats per minute and blood pressure is 126/72 mmHg. His chest is clear, abdomen soft and non-tender and there is no focal neurology.

Repeat blood tests and arterial blood gas are taken.

Which electrolyte abnormality is most likely to have caused his seizure?

○ Hypocalcaemia ✕

○ Hypoglycaemia ✕

○ Hypokalaemia ✕

○ Hypomagnesaemia ✕

○ Hypophosphataemia ✕

Submit answer

Reference ranges

Score: **50.7%**

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Repeat blood tests and arterial blood gas are taken.

Which electrolyte abnormality is most likely to have caused his seizure?

- ○ Hypocalcaemia ×
- ○ Hypoglycaemia ×
- ○ Hypokalaemia ×

○ Hypomagnesaemia ✗

○ Hypophosphataemia ✗

Submit answer

Reference ranges ⌄

Score: **50.7%**

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Repeat blood tests and arterial blood gas are taken.

Which electrolyte abnormality is most likely to have caused his seizure?

Hypocalcaemia	10%
Hypoglycaemia	20%
Hypokalaemia	3%

Hypomagnesaemia	28%
Hypophosphataemia	39%

Hypophosphataemia is the most common electrolyte abnormality during alcohol withdrawal and is a recognised cause of seizures. It is often present at baseline in alcoholic patients and can further decrease in withdrawal. It is thought that this is due to effects on the proximal renal tubule. It can cause muscle pain and disorientation at very low levels (< 0.3 mmol/l).

Hypoglycaemia would also be possible but in the absence of diabetes, and with the patient eating and drinking, this is less likely. Hypoglycaemia is common in patients intoxicated with alcohol and these patients should be monitored with regular capillary blood glucose testing.

Low magnesium and low calcium both cause seizures but are unlikely to be lowered sufficiently in this patient to result in seizures in the absence of other clinical signs.

Low potassium is unlikely to result in seizures.

References:
Vandermergel & Simon. Evolution of Metabolic Abnormalities in Alcoholic Patients during Withdrawal. Journal of addiction. 2015.
Castilla-Guerra et al. Electrolyte disturbances and seizures. Epilepsia. 2006.

👍 👎 Discuss (8) Improve

Next question

Hypophosphataemia

Causes

- alcohol excess

- acute liver failure
- diabetic ketoacidosis
- refeeding syndrome
- primary hyperparathyroidism
- osteomalacia

Consequences

- red blood cell haemolysis
- white blood cell and platelet dysfunction
- muscle weakness and rhabdomyolysis
- central nervous system dysfunction

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypophosphataemia

Osmosis - YouTube

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Report broken media

Score: **51.3%**

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Question 77 of 217

A 26-year-old woman presents to her GP with a 6-month history of excessive thirst and passing large volumes of urine, particularly during the day. She drinks approximately 6 litres of water daily and reports no significant past medical history or medication use. She is otherwise well, with a normal BMI and unremarkable physical examination. Initial blood tests, including serum electrolytes and glucose, are within normal limits. To further evaluate her symptoms, a water deprivation test is performed:

Test	Value	Reference Range
Plasma osmolality (start)	270 mOsm/kg	(275–295)
Urine osmolality after deprivation	430 mOsm/kg	(>400)
Urine osmolality after desmopressin	445 mOsm/kg	(>400)

Which is the most likely diagnosis?

- ○ Primary polydipsia ✕
- ○ Cranial diabetes insipidus ✕
- ○ Nephrogenic diabetes insipidus ✕
- ○ Partial cranial diabetes insipidus ✕
- ○ Type 2 diabetes mellitus ✕

Submit answer

Reference ranges

Score: **51.3%**

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Urine osmolality after desmopressin	445 mOsm/kg	(>400)

Which is the most likely diagnosis?

🟩	Primary polydipsia	68%
	Cranial diabetes insipidus	6%
🟥	Nephrogenic diabetes insipidus	24%
	Partial cranial diabetes insipidus	3%
	Type 2 diabetes mellitus	0%

Water deprivation test: primary polydipsia

- urine osmolality after fluid deprivation: high
- urine osmolality after desmopressin: high

Important for me Less important

Primary polydipsia is the correct answer because this condition typically presents in young adults with excessive water intake resulting in polyuria. The key distinguishing feature on water deprivation testing is that both urine osmolality after deprivation and after desmopressin administration are appropriately raised (>400 mOsm/kg), indicating intact renal concentrating ability—consistent with psychogenic or primary polydipsia. The starting plasma osmolality is low due to chronic overhydration, further supporting this diagnosis. Recognition of these findings prevents unnecessary interventions for other causes of polyuria.

Cranial diabetes insipidus would present with an inability to concentrate urine following fluid deprivation—urine osmolality remains low (<300 mOsm/kg)—but responds significantly to desmopressin (urine osmolality rises sharply). In this scenario, the patient's urine concentrates even before desmopressin administration, making cranial DI unlikely.

Nephrogenic diabetes insipidus involves renal resistance to ADH; thus, both after fluid deprivation and after desmopressin administration, urine remains dilute (<300 mOsm/kg). Here, the patient's kidneys respond normally by concentrating urine above this threshold during both phases, which rules out nephrogenic DI.

Partial cranial diabetes insipidus may show some increase in urine osmolality post-desmopressin but not as robustly as seen here—the patient’s results suggest normal endogenous ADH function rather than partial deficiency.

Type 2 diabetes mellitus can cause osmotic diuresis if glucose levels are high enough to exceed the renal threshold; however, initial bloods were normal and there was no mention of hyperglycaemia or glucosuria. Furthermore, type 2 DM does not affect urinary concentrating ability in this manner.

👍 👎 💬 Discuss Improve

Next question

Water deprivation test

The water deprivation test is designed to help evaluate patients who have polydipsia.

Method
- prevent patient drinking water
- ask the patient to empty their bladder
- hourly urine and plasma osmolalities

	Starting plasma osm.	**Final urine osm.**	**Urine osm. post-DDAVP**
Normal	Normal	> 600	> 600
Psychogenic polydipsia	Low	> 400	> 400
Cranial DI	High	< 300	> 600
Nephrogenic DI	High	< 300	< 300

+ Q 123

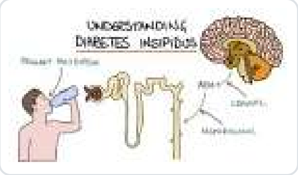
Next question

Textbooks

High-yield textbook

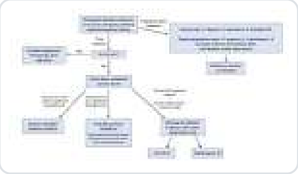
Extended textbook

Media


UNDERSTANDING
DIABETES INSIPIDUS
ADH

Understanding Diabetes Insipidus

Zero to Finals - YouTube 👍 3 👎 0



An Approach to Polyuria

Strong Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **50.6%**

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A 45-year-old female presents to the clinic with right sided flank pain. A CT KUB confirms a right sided kidney stone. She has a past medical history of asthma, rheumatoid arthritis and Sjogren's syndrome. Her drug history includes salbutamol, methotrexate and hydroxychloroquine.

Blood results are as follows:

Hb	115 g/l	Na$^+$	136 mmol/l
Platelets	460 * 10^9/l	K$^+$	3.1 mmol/l
Bicarbonate	16 mEq/L (normal 22-28)	Urea	7.8 mmol/l
Chloride	115mmol/l	Creatinine	76 µmol/l

How would you prevent further kidney stones?

○ Thiazide diuretics ×

○ Loop diuretics ×

○ Penicillamine ×

○ Encourage increased fluid intake ×

○ Sodium bicarbonate ×

Submit answer

Reference ranges

Score: **50.6%**

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Question 78 of 217 ✓

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Bicarbonate	16 mEq/L (normal 22-28)	Urea	7.8 mmol/l
Chloride	115mmol/l	Creatinine	76 µmol/l

How would you prevent further kidney stones?

Thiazide diuretics	48%
Loop diuretics	4%
Penicillamine	4%
Encourage increased fluid intake	14%
Sodium bicarbonate	30%

Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate

Important for me Less important

The patient has a low bicarbonate which confirms a metabolic acidosis. This should prompt you to work out the anion gap which can be extremely useful diagnostically.

The anion gap in this example = sum of positive cations - sum of negative anions = (Na + K) - (HCO3 + Cl) = 9.1 We are therefore dealing with a normal anion gap metabolic acidosis (NAGMA).

High anion gap metabolic acidosis (HAGMA) is generally caused by the following conditions which can be remembered with the mnemonic KULT

- Ketoacidosis: diabetic, starvation or alcoholic
- Uraemia
- Lactic acidosis
- Toxins e.g. salicylates, ethylene glycol, methanol

Normal anion gap metabolic acidosis (NAGMA) is generally caused by the following conditions which can be remembered with the mnemonic ABCD

- Addison's disease
- Bicarbonate loss via GI tract (e.g. diarrhoea) or renal tract (e.g. renal tubular acidosis)
- Chloride excess - hyperchloraemic acidosis
- Drugs e.g. acetazolamide

In this case, the NAGMA, hypokalaemia, and renal stones should prompt you to consider renal tubular acidosis (RTA). The presence of kidney stones, and the past medical history of Sjogren's syndrome and rheumatoid arthritis make RTA type 1 (distal) the most likely diagnosis. Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate.

👍 👎 💬 Discuss (7) Improve

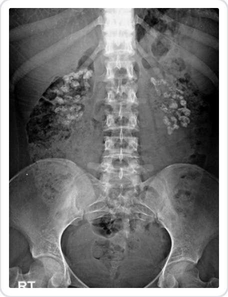
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

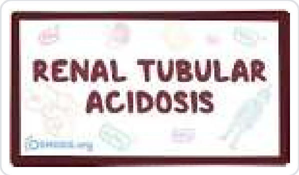
Next question

Textbooks

High-yield textbook

Extended textbook

Media


RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **51.3%**

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A 50-year-old woman with a history of Grave's disease is reviewed on the surgical ward some 12hrs after parathyroidectomy. She has begun suffering from episodes of carpopedal spasm and pins and needles affecting both hands and around her mouth. On examination on the ward, her blood pressure is 115/72 mmHg, and pulse is 88 beats per minute. Her serum calcium is measured at 1.85 mmol/l.

Which of the following is the most appropriate intervention?

○ Intravenous diazepam ✕

○ Intravenous calcium ✕

○ Intravenous magnesium ✕

○ Oral calcium ✕

○ Oral vitamin D ✕

Submit answer

Reference ranges

Score: **51.3%**

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Question 79 of 217 ✓

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Which of the following is the most appropriate intervention?

Intravenous diazepam	1%
[✓ Intravenous calcium]	89%
Intravenous magnesium	6%
Oral calcium	4%
Oral vitamin D	1%

Intravenous calcium gluconate is used for the acute management of hypocalcaemia

Important for me Less important

This patient has symptomatic hypocalcaemia, most likely due to an acute fall in parathyroid hormone after surgery. This is considered a medical emergency and calcium replacement IV is essential:

IV calcium gluconate is administered initially with 20 ml of 10% calcium gluconate in 50-100 ml of 5% dextrose IV, given over 10 minutes with ECG monitoring.This can be repeated until the patient is asymptomatic. It should be followed up with a calcium gluconate infusion where 100ml of 10% calcium gluconate is diluted in 1 litre of normal saline or 5% dextrose and infused at 50-100 ml/hr.

Not intervening with respect to the electrolyte disturbance risks significant sequelae including cardiac arrhythmia, diazepam is therefore not appropriate. IV magnesium is most useful where hypocalcaemia is resistant to correction, and oral interventions would take too long to elevate serum calcium levels.

👍 👎 Discuss (6) Improve

Next question

Hypocalcaemia: causes and management ★

The clinical history combined with parathyroid hormone levels will reveal the cause of hypocalcaemia in the majority of cases

Causes

- vitamin D deficiency (osteomalacia)
- chronic kidney disease
- hypoparathyroidism (e.g. post thyroid/parathyroid surgery)
- pseudohypoparathyroidism (target cells insensitive to PTH)
- rhabdomyolysis (initial stages)
- magnesium deficiency (due to end organ PTH resistance)
- massive blood transfusion
- acute pancreatitis

Contamination of blood samples with EDTA may also give falsely low calcium levels.

Management

- severe hypocalcaemia (e.g. carpopedal spasm, tetany, seizures or prolonged QT interval) requires IV calcium replacement
    - the preferred method is with intravenous calcium gluconate, 10ml of 10% solution over 10 minutes
    - intravenous calcium chloride is more likely to cause local irritation
    - ECG monitoring is recommended

- further management depends on the underlying cause

+ Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 3 👎 2

Report broken media

Score: **51.9%**

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A 73-year-old woman presents to the general medical clinic. She has been investigated for hyponatraemia and has come for discussion of her results. She was incidentally found to have low sodium after having blood tests during a community-acquired chest infection two weeks ago. The chest infection has entirely resolved. She was found to have radiological evidence of small cell lung cancer on CT scan of her chest during investigations for her chest infection as well. She declined further biopsy or any investigation for this finding. Clinically she is euvolaemic. She has had serum sodium and osmolality measured. She is suspected of having SIADH.

Na^+	122 mmol/l
K^+	4.1 mmol/l
Urea	4.8 mmol/l
Creatinine	69 µmol/l
Serum osmolality	240 mmol/kg

What further investigation would be necessary to diagnose SIADH?

- ○ Random serum cortisol ×
- ○ Echocardiogram ×
- ○ Lung biopsy ×
- ○ Urinary electrolytes and osmolality ×
- ○ Water deprivation test ×

Submit answer

Reference ranges

Score: **51.9%**

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Question 80 of 217 ✓

A 73-year-old woman presents to the general medical clinic. She has been investigated for hyponatraemia and has come for discussion of her results. She was incidentally found to have low sodium after having blood tests during a community-acquired chest infection two weeks ago. The chest infection has entirely resolved. She was found to have radiological evidence of small cell lung cancer on CT scan of her chest during investigations for her chest infection as well. She declined further biopsy or any investigation for this finding. Clinically she is euvolaemic. She has had serum sodium and osmolality measured. She is suspected of having SIADH.

Na^+	122 mmol/l
K^+	4.1 mmol/l
Urea	4.8 mmol/l
Creatinine	69 µmol/l
Serum osmolality	240 mmol/kg

What further investigation would be necessary to diagnose SIADH?

Random serum cortisol	2%
Echocardiogram	0%
Lung biopsy	1%
Urinary electrolytes and osmolality	86%
Water deprivation test	10%

SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient

Important for me Less important

This patient has established hyponatraemia with low serum osmolality. She needs to have elevated urinary osmolality to confirm the diagnosis of SIADH in the context of hyponatraemia and no other cause such as hypervolaemia. A morning cortisol would be helpful in diagnosing Addison's disease, and if suspected would be a useful investigation, but a random serum cortisol would not be very helpful. An echocardiogram could exclude heart failure in the context of fluid overload as a cause of hyponatraemia, but in the absence of peripheral oedema, a raised JVP and pulmonary oedema this is unlikely to be necessary. Water deprivation test is done to confirm diabetes insipidus.

Discuss (2) Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology

- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water balance
- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels
- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.

- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension, because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted, leading to hyponatraemia.

Causes of SIADH

Category	**Examples**
Malignancy	**small cell lung cancer** also: pancreas, prostate
Neurological	stroke subarachnoid haemorrhage subdural haemorrhage meningitis/encephalitis/abscess
Infections	tuberculosis pneumonia
Drugs	sulfonylureas* SSRIs, tricyclics carbamazepine vincristine cyclophosphamide
Other causes	positive end-expiratory pressure (PEEP) porphyrias

Investigations

- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.

- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management

- correction must be done slowly to avoid precipitating central pontine myelinolysis
- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **52.5%**

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A 46-year-old man visits his general practitioner with an 8-week history of reduced libido. He has never experienced this before and is becoming very distressed. There is no history of recent illness or change in weight and he denies any headaches or visual symptoms.

On examination, his observations are taken which are all within normal range. He has a soft and non-tender abdomen with no stigmata of liver disease. His testicular examination is unremarkable and there is no evidence of scrotal swelling. There is a visible milky discharge from both nipples.

What is the most likely cause of this patient's presentation?

- ○ Carbamazepine ✕
- ○ Lithium ✕
- ○ Metoclopramide ✕
- ○ Ropinirole ✕
- ○ Spironolactone ✕

Submit answer

Reference ranges

Score: **52.5%**

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Question 81 of 217 ✓

A 46-year-old man visits his general practitioner with an 8-week history of reduced libido. He has never experienced this before and is becoming very distressed. There is no history of recent illness or change in weight and he denies any headaches or visual symptoms.

On examination, his observations are taken which are all within normal range. He has a soft and non-tender abdomen with no stigmata of liver disease. His testicular examination is unremarkable and there is no evidence of scrotal swelling. There is a visible milky discharge from both nipples.

What is the most likely cause of this patient's presentation?

Carbamazepine	2%
Lithium	3%
Metoclopramide	67%
Ropinirole	6%
Spironolactone	22%

Metoclopramide may result in galactorrhoea

Important for me Less important

This patient has reduced libido and galactorrhoea secondary to increased prolactin levels. Other features of raised prolactin levels include impotence in men and amenorrhoea in women. There are multiple causes of raised prolactin levels such as prolactinoma, PCOS, hypothyroidism and medication use. Dopamine inhibits the release of prolactin from the anterior pituitary gland and therefore drugs that decrease dopamine levels increase the amount of prolactin release. Of the drugs given in the question, metoclopramide is the only dopamine antagonist. It is a D2 and serotonin 5-HT3 receptor antagonist in the chemoreceptor trigger zone.

Carbamazepine is an anti-convulsant medication. Although it's mechanism of action is still widely debated, it is thought that it works by inhibiting sodium channels. Carbamazepine has multiple side effects including dizziness, drowsiness, hyponatraemia and thrombocytopenia. In rare cases, it can cause gynaecomastia. However, it does not cause galactorrhoea.

Lithium is a mood stabiliser used in the treatment of mania, bipolar disorder and recurrent depression. Lithium has multiple potential side effects including alopecia, arrhythmias, dizziness, seizures and coma. It does not cause galactorrhoea.

Ropinirole is a dopamine agonist used in the treatment of Parkinson's disease. Raised dopamine levels would decrease the amount of prolactin and, therefore, dopamine agonists are used to control galactorrhoea.

Spironolactone is an aldosterone antagonist. A side effect of spironolactone that is commonly tested in exam questions is the development of gynaecomastia. However, galactorrhoea is not seen.

👍 👎 Discuss (3) Improve

Next question

Prolactin and galactorrhoea ★

Prolactin is secreted by the anterior pituitary gland with release being controlled by a wide variety of physiological factors. Dopamine acts as the primary prolactin releasing inhibitory factor and hence dopamine agonists such as bromocriptine may be used to control galactorrhoea. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia

Features of excess prolactin

- men: impotence, loss of libido, galactorrhoea
- women: amenorrhoea, galactorrhoea

Causes of raised prolactin

- prolactinoma
- pregnancy
- oestrogens
- physiological: stress, exercise, sleep
- acromegaly: 1/3 of patients
- polycystic ovarian syndrome
- primary hypothyroidism (due to thyrotrophin releasing hormone (TRH) stimulating prolactin release)

Drug causes of raised prolactin

- metoclopramide, domperidone
- phenothiazines
- haloperidol
- very rare: SSRIs, opioids

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 11 👎 5

Galactorrhoea review

Suggest link Report broken link

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **53.1%**

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PassMedicine

← Question 82 of 217 →

You review a 28-year-old woman who is 26 weeks pregnant. She has just had a routine oral glucose tolerance test as her BMI is 34 kg/m². The following results were obtained:

Time (hours)	Blood glucose (mmol/l)
0	7.4
2	11.2

There have been no other antenatal problems and her anomaly scan was normal. What is the most appropriate action?

- ○ Repeat oral glucose tolerance test in 4 weeks ×
- ○ Start metformin + advice about diet / exercise + self-monitor glucose levels ×
- ○ Advice about diet / exercise + self-monitor glucose levels ×
- ○ Start insulin + advice about diet / exercise + self-monitor glucose levels ×
- ○ Reassure results within normal limits ×

Submit answer

Reference ranges

Score: **53.1%**

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Time (hours)	**Blood glucose (mmol/l)**
0	7.4
2	11.2

There have been no other antenatal problems and her anomaly scan was normal. What is the most appropriate action?

Repeat oral glucose tolerance test in 4 weeks	3%
Start metformin + advice about diet / exercise + self-monitor glucose levels	34%
Advice about diet / exercise + self-monitor glucose levels	16%
Start insulin + advice about diet / exercise + self-monitor glucose levels	45%
Reassure results within normal limits	2%

NICE have recently changed their gestational diabetes guidelines. Insulin should be started in the fasting glucose is >= 7 mmol/l. Aspirin should also be considered given the increased risk of pre-eclampsia.

Discuss (5) | Improve

Next question

Gestational diabetes

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:

- 87.5% have gestational diabetes
- 7.5% have type 1 diabetes
- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes

- BMI of > 30 kg/m^2
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes
- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)
- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:

- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose
- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
    - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
    - if glucose targets are still not met insulin should be added to diet/exercise/metformin
    - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios, insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets with metformin but decline insulin treatment

Management of pre-existing diabetes

- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	Target
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link

Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

Report broken media

Score: **53.7%**

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Question 83 of 217

A 47-year-old woman has attended the endocrinology clinic for a review of hypothyroidism. She has been well maintained on 75micrograms of levothyroxine, once daily, for the past 4 years but describes worsening symptoms over the last 6 weeks.

She describes fatigue, low mood, constipation, and weight gain but is adamant she has not missed any doses of her medication.

She explains that she has recently been commenced on a few new medications by her GP including citalopram, ferrous fumarate, ramipril, propranolol, and metformin. All of her medications are taken in the morning.

Thyroid function tests today:

Thyroid-stimulating hormone (TSH)	12.4 mU/L	(0.5-5.5)
Free thyroxine (T4)	3.6 pmol/L	(9.0 - 18)

Thyroid function tests 3 months ago:

Thyroid-stimulating hormone (TSH)	2.6 mU/L	(0.5-5.5)
Free thyroxine (T4)	14.1 pmol/L	(9.0 - 18)

Which of her medications would explain her blood test results?

- ○ Citalopram ×
- ○ Ferrous fumarate ×
- ○ Metformin ×
- ○ Propranolol ×

○ Ramipril ×

Submit answer

Reference ranges ⌄

Score: **53.7%**

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Free thyroxine (T4)	3.6 pmol/L	(9.0 - 18)

Thyroid function tests 3 months ago:

Thyroid-stimulating hormone (TSH)	2.6 mU/L	(0.5-5.5)
Free thyroxine (T4)	14.1 pmol/L	(9.0 - 18)

Which of her medications would explain her blood test results?

Citalopram	9%
[✓ Ferrous fumarate]	86%
Metformin	1%
Propranolol	4%

Ramipril 0%

Iron / calcium carbonate tablets can reduce the absorption of levothyroxine - should be given 4 hours apart

Important for me Less important

Ferrous fumarate is the correct answer. Iron tablets commonly affect levothyroxine absorption which can lead to symptomatic hypothyroidism. It is best to leave a 4-hour gap between consuming iron and levothyroxine.

Citalopram does not have any interactions with levothyroxine.

Metformin does not affect levothyroxine, however, levothyroxine can interfere with blood glucose and affect the ability of metformin.

Propranolol does not interact with levothyroxine.

Ramipril does not have any proven interactions with levothyroxine, however, studies are currently ongoing.

👍 👎 💬 Discuss (1) Improve

Next question

Hypothyroidism: levothyroxine therapy ★

Key points

- initial starting dose of levothyroxine should be lower in elderly patients and those with ischaemic heart disease. The BNF recommends that for patients with cardiac disease, severe hypothyroidism or patients over 50 years the initial starting dose should be 25mcg od with dose slowly titrated. Other patients should be started on a dose of 50-100mcg od
- following a change in thyroxine dose thyroid function tests should be checked after 8-12 weeks

- the therapeutic goal is 'normalisation' of the thyroid stimulating hormone (TSH) level. As the majority of unaffected people have a TSH value 0.5-2.5 mU/l it is now thought preferable to aim for a TSH in this range
- women with established hypothyroidism who become pregnant should have their dose increased by at least 25-50 micrograms levothyroxine due to the increased demands of pregnancy. The TSH should be monitored carefully, aiming for a low-normal value
- there is no evidence to support combination therapy with levothyroxine and liothyronine

Side-effects of thyroxine therapy

- hyperthyroidism: due to over treatment
- reduced bone mineral density
- worsening of angina
- atrial fibrillation

Interactions

- iron, calcium carbonate
    - absorption of levothyroxine reduced, give at least 4 hours apart

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 15 👎 24

Management of hypothyroidism

Suggest link Report broken link

Media

HYPOTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hypothyroidism treatment medications

Osmosis - YouTube 👍 3 👎 1

Report broken media

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Question 84 of 217

A 47-year-old woman is admitted to the surgical ward with severe loin to groin abdominal pain. A CT-KUB reveals a right-sided renal calculus. When you clerk her in she admits to you that she has not felt herself for the past few weeks with polyuria, polydipsia, constipation and altered mood.

Blood tests show:

Estimated glomerular filtration rate	>60 ml/min
Adjusted calcium	3.1 mmol/l (2.1-2.6 mmol/l)
Phosphate	0.6 mmol/l (0.8-1.4 mol/l)
Parathyroid hormone	5.1 pmol/l (1.2-5.8 pmol/l)

Which of the following is the most likely cause for her symptoms?

- ○ Primary hyperparathyroidism ✕
- ○ Secondary hyperparathyroidism ✕
- ○ Sarcoidosis ✕
- ○ Tertiary hyperparathyroidism ✕
- ○ Type 1 renal tubular acidosis ✕

Submit answer

Reference ranges

Score: **54.2%**

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Question 84 of 217 ✓

A 47-year-old woman is admitted to the surgical ward with severe loin to groin abdominal pain. A CT-KUB reveals a right-sided renal calculus. When you clerk her in she admits to you that she has not felt herself for the past few weeks with polyuria, polydipsia, constipation and altered mood.

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Phosphate	0.6 mmol/l (0.8-1.4 mol/l)
Parathyroid hormone	5.1 pmol/l (1.2-5.8 pmol/l)

Which of the following is the most likely cause for her symptoms?

Primary hyperparathyroidism	71%
Secondary hyperparathyroidism	6%
Sarcoidosis	4%
Tertiary hyperparathyroidism	4%
Type 1 renal tubular acidosis	16%

The PTH level in primary hyperparathyroidism may be normal

Important for me Less important

The most likely diagnosis here is primary hyperparathyroidism caused by parathyroid adenoma or hyperplasia. The classical biochemical findings are a high serum calcium and low phosphate. The parathyroid hormone level is either high or inappropriately normal.

Secondary hyperparathyroidism is caused by chronic hypocalcaemia (e.g. chronic kidney disease). Serum calcium is low or normal whilst parathyroid hormone levels are high.

Tertiary hyperparathyroidism develops from secondary hyperparathyroidism and results in autonomous parathyroid production. It is usually seen patients with end-stage renal disease.

Sarcoidosis and type 1 renal tubular acidosis are rare causes of hypercalcaemia.

👍 👎 💬 Discuss (5) Improve

Next question

Primary hyperparathyroidism ★

Primary hyperparathyroidism is caused by excess secretion of PTH resulting in hypercalcaemia. It is the most common cause of hypercalcaemia in outpatients and is often diagnosed following an incidental finding of an elevated serum calcium concentration. In 85% of cases a parathyroid adenoma is responsible.

Causes of primary hyperparathyroidism
- 85%: solitary adenoma
- 10%: hyperplasia
- 4%: multiple adenoma
- 1%: carcinoma

Around 80% of patients are asymptomatic and are diagnosed on routine blood tests. The symptomatic features of primary hyperparathyroidism may be remembered by the mnemonic: 'bones, stones, abdominal groans and psychic moans':
- polydipsia, polyuria
- depression
- anorexia, nausea, constipation

- peptic ulceration
- pancreatitis
- bone pain/fracture
- renal stones
- hypertension

Associations

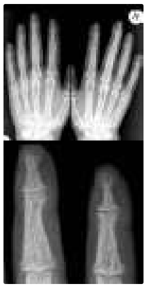
- hypertension
- multiple endocrine neoplasia: MEN I and II

Investigations

- bloods
  - raised calcium, low phosphate
  - PTH may be raised or (inappropriately, given the raised calcium) normal
- technetium-MIBI subtraction scan
- x-ray findings
  - pepperpot skull
  - osteitis fibrosa cystica

Treatment

- the definitive management is total parathyroidectomy
- conservative management may be offered if the calcium level is less than 0.25 mmol/L above the upper limit of normal AND the patient is > 50 years AND there is no evidence of end-organ damage
- patients not suitable for surgery may be treated with cinacalcet, a calcimimetic
  - a calcimimetic 'mimics' the action of calcium on tissues by allosteric activation of the calcium-sensing receptor



+ Q 123

Next question

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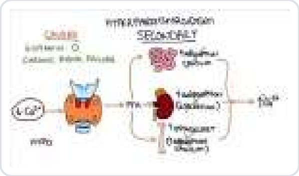
Media


HYPERPARATHYROIDISM
SECONDARY

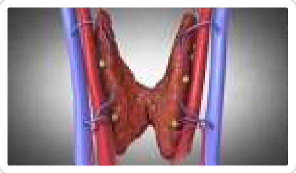
Understanding Hyperparathyroidism

Zero to Finals - YouTube 👍 13 👎 0

ENDOCRINOLOGY
HYPERPARATHYROIDISM

Hyperparathyroidism and the different types, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 2 👎 0



[Parathyroid Glands and Hyperparathyroidism]

Norman Parathyroid Center - YouTube 👍 2 👎 1

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Question 85 of 217

A 25-year-old woman is referred urgently to the endocrinology clinic. She became pregnant three weeks ago. She had been suffering from diarrhea for the last month, and despite taking the oral contraceptive pill, became pregnant. She went to see her GP who requested blood tests as well as organizing an antenatal referral. Her full blood count, renal profile and liver function tests are all normal, but her TSH is undetectable and her free T4 levels are 52pmol/l. By the time of referral she has also had thyroid-stimulating hormone receptor antibodies which are positive. She has no past medical history. What is the most appropriate management for her hyperthyroidism?

- ○ Hemithyroidectomy ✕
- ○ Carbimazole ✕
- ○ Propylthiouracil ✕
- ○ Propranolol ✕
- ○ Total thyroidectomy ✕

Submit answer

Reference ranges

Score: **54.8%**

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Question 85 of 217 ✓

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	Hemithyroidectomy	0%
	Carbimazole	7%
✓	Propylthiouracil	89%
	Propranolol	3%
	Total thyroidectomy	0%

In pregnant woman who develop hyperthyroidism in the first trimester, propylthiouracil is preferred over carbimazole due to lower risk of foetal malformation

Important for me Less important

This patient has a new diagnosis of Graves' disease with positive thyroid-stimulating hormone receptor antibodies whilst pregnant in the first trimester. The most appropriate medical treatment is propylthiouracil. This has a lower risk of foetal malformation than carbimazole in the first trimester and would, therefore, be first-line treatment. Radioiodine is contra-indicated in pregnancy, whilst propranolol would not address the underlying problem. Surgery can be an option to patients who are resistant to propylthiouracil, and would need to happy at an urgent basis. Of operations, a hemithyroidectomy is more appropriate in trying to obtain histological samples in suspicious histology, whilst a total thyroidectomy would be a definitive management. Urgent treatment is necessary as thyroid-stimulating hormone receptor antibodies cross the placenta and can cause foetal problems.

Discuss (6) | Improve

Next question

Pregnancy: thyroid problems

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism

- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points

- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link

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A 27-year-old female presents with secondary amenorrhoea after stopping the oral contraceptive pill 6 months ago. She gets regular headaches and struggles to stand from seated or climb stairs.

On examination, milk could be expressed from the breasts and visual fields showed bilateral defects in the upper outer quadrants.

Prolactin	1080 mIU/L (NR<360)
FSH	0.1 IU/L (NR 1-11)
LH	0.2 IU/L (NR 20-75)
TSH	0.1 mIU/L (NR 0.3-6.0)
T4	8 pmol/L (NR 10-25)
9am cortisol	20 nmol/L (NR 140-700)

Pituitary MRI: 3cm pituitary mass with tenting of optic chiasm.

What is the next step in management?

- ○ Bromocriptine ×
- ○ Octreotide ×
- ○ Stereotactic radiotherapy ×
- ○ Trans-sphenoidal surgery ×
- ○ Transcranial hypophysectomy ×

Submit answer

Reference ranges

Score: **55.3%**

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TSH	0.1 mIU/L (NR 0.3-6.0)
T4	8 pmol/L (NR 10-25)
9am cortisol	20 nmol/L (NR 140-700)

Pituitary MRI: 3cm pituitary mass with tenting of optic chiasm.

What is the next step in management?

Bromocriptine	48%
Octreotide	5%
Stereotactic radiotherapy	1%
Trans-sphenoidal surgery	46%
Transcranial hypophysectomy	1%

This patient has a macroadenoma (>1cm) causing visual field defects. Trans-sphenoidal surgery is the first step in management. Raised prolactin can be secondary to blockage of the pituitary stalk with prevention of dopamine reaching the pituitary causing disinhibition of the lactotrophs.

Prolactin secreting macroadenomas secrete very high quantities and PRL is usually >6000mU/ml particularly with macro-prolactinomas (this is not the case making a prolactinoma unlikely). Prolactinomas are treated with dopamine agonists (bromocriptine, cabergoline) first-line. Octreotide is used to treat acromegaly.

Transcranial hypophysectomy is done for very large tumours that cant be removed via the trans-sphenoidal route.

👍 👎 Discuss (11) Improve

Next question

Pituitary adenoma ★

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:

- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:

- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due to excess prolactin)

- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
    - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
    - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).
    - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
    - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone) and neuromodulators like pasireotide

- transsphenoidal surgery
    - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
    - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
    - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
    - indicated for residual or recurrent tumours post-surgery

AI + Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 20 👎 11

Pituitary tumours review

Suggest link Report broken link

Score: **55.8%**

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A 35-year-old woman is referred to the endocrinology clinic after having lost 5kg without any change in diet or exercise habits. On direct questioning, she also describes that she has noticed feeling warmer, losing sleep, and a slight tremor in her hands. She has a past medical history of depression for which she takes sertraline. She has no other medical problems. On examination, she has a body mass index of 24 kg/m², a slight tremor at rest, and a large goitre. This is non-tender and there are no palpable nodules. There is no lymphoadenopathy present. Blood tests show a normal full blood count and electrolytes, but TSH is undetectable and free T4 is 39pmol/l. She is clinically diagnosed with hyperthyroidism. What further investigation is most appropriate to determine the type of her hyperthyroidism?

- ○ Thyroid-stimulating hormone receptor antibodies ×
- ○ Liver function tests ×
- ○ CT neck ×
- ○ Scan of radionuclide thyroid uptake ×
- ○ US-guided fine needle aspiration cytology ×

Submit answer

Reference ranges

Score: **55.8%**

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Back to top

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Thyroid-stimulating hormone receptor antibodies	56%
Liver function tests	0%
CT neck	1%
Scan of radionuclide thyroid uptake	42%
US-guided fine needle aspiration cytology	1%

TSH antibodies are found in 90% of patients with Graves' disease and can help distinguish from other forms of hyperthyroidism

Important for me Less important

This patient clinically and biochemically has hyperthyroidism. To help determine the cause of hyperthyroidism, and distinguish between causes such as Graves' disease, toxic adenoma and toxic multinodular goitre. Thyroid-stimulating hormone receptor antibodies (TSH-RAb, commonly shortened to TRAb) has an up to 98% sensitivity and 99% specificity for Graves' disease. If this test is negative, then a radionuclide thyroid uptake scan can help determine the cause. In Graves' disease there is diffuse high uptake, in thyroiditis there is low uptake, and if there are nodules then there is uneven uptake. US-guided fine needle aspiration cytology is recommended in the presence of features suggestive of malignancy. Liver function tests are important if there is a history of liver disease as carbimazole can cause further liver injury, but it would not help in determining the cause of hyperthyroidism.

Discuss (12) Improve

Next question

Graves' disease: features

Graves' disease is an autoimmune thyroid disease in which the body produces IgG antibodies to the thyroid-stimulating hormone (TSH) receptor. It is the most common cause of thyrotoxicosis and is typically seen in women aged 30-50 years.

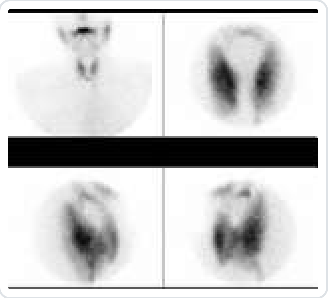
Features
- typical features of thyrotoxicosis
- specific signs limited to Grave's (see below)

Features seen in Graves' but not in other causes of thyrotoxicosis
- eye signs (30% of patients)
    - exophthalmos
    - ophthalmoplegia
- pretibial myxoedema
- thyroid acropachy, a triad of:
    - digital clubbing
    - soft tissue swelling of the hands and feet
    - periosteal new bone formation

Autoantibodies
- TSH receptor stimulating antibodies (90%)
- anti-thyroid peroxidase antibodies (75%)

Thyroid scintigraphy

- diffuse, homogenous, increased uptake of radioactive iodine



Q 123

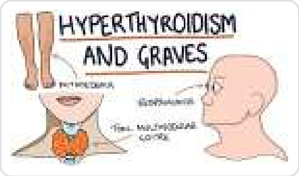
Next question

Textbooks

High-yield textbook

Extended textbook

Media


HYPERTHYROIDISM
AND GRAVES

[Understanding Hyperthyroidism and Graves' Disease]

Zero to Finals - YouTube

👍 2 👎 0

ENDOCRINOLOGY
GRAVES DISEASE

Graves' disease

Armando Hasudungan - YouTube 👍 1 👎 1

Report broken media

Score: **56.3%**

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A 20-year-old female admits to abusing codeine and diclofenac, up to 30 tablets per day. She attends the emergency department demanding help with her addiction. Her baseline bloods include:

pH	7.28
PCO2	2.9 kPa
PO2	8.5 kPa
HCO3-	16 mmol/l

Na+	130 mmol/l
K+	6 mmol/l
Cl-	110 mmol/l
HCO3-	16 mmol/l

What is the most likely diagnosis?

- ○ Lactic acidosis
- ○ Type 1 renal tubular acidosis
- ○ Type 2 renal tubular acidosis
- ○ Type 4 renal tubular acidosis
- ○ Ketoacidosis

Submit answer

Reference ranges

Score: **56.3%**

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Question 88 of 217 ✓

A 20-year-old female admits to abusing codeine and diclofenac, up to 30 tablets per day. She attends the emergency department demanding help with her addiction. Her baseline bloods include:

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PO2	8.5 kPa
HCO3-	16 mmol/l

Na+	130 mmol/l
K+	6 mmol/l
Cl-	110 mmol/l
HCO3-	16 mmol/l

What is the most likely diagnosis?

Lactic acidosis	4%
Type 1 renal tubular acidosis	10%
Type 2 renal tubular acidosis	7%
Type 4 renal tubular acidosis	78%
Ketoacidosis	0%

Type 4 renal tubular acidosis is due either to a deficiency of aldosterone or to a resistance to its effects.

Causes include:

- Aldosterone deficiency (hypoaldosteronism): Primary vs. hyporeninaemic

- Aldosterone resistance
- → 1.Drugs: Non-steroidal anti-inflammatories, angiotensin converting enzyme inhibitors, angiotensin 2 receptor blockers, eplerenone, spironolactone, trimethoprim, pentamidine
- → 2.Pseudohypoaldosteronism

👍 👎 Discuss (7) Improve

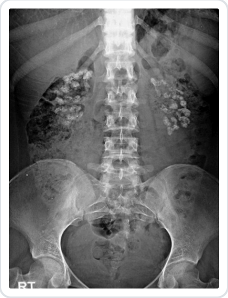
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase

inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)
- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)
- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Dr. Jamil Alrajhi

Textbooks

High-yield textbook

Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **56.8%**

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A 45-year-old gentleman presents to clinic for review. Two weeks ago he presented to the emergency department with renal colic. A spiral CT KUB confirmed nephrolithiasis and he was managed conservatively with IV fluids, analgesia and an alpha-blocker. His symptoms resolved entirely and he was discharged.

Blood tests:

Hb	142 g/l
Platelets	329 * 10^9/l
WBC	6.6 * 10^9/l
Na^+	141 mmol/l
K^+	3.8 mmol/l
Urea	6.2 mmol/l
Creatinine	71 µmol/l
Corrected calcium	2.71 mmol/l
Parathyroid hormone	10.2 pmol/l (1.0-7.0 pmol/l)

How should he be further managed?

- ○ Annual monitoring of calcium and renal function ✕
- ○ Encourage oral fluids ✕
- ○ Bisphosphonates ✕
- ○ Vitamin D supplementation ✕
- ○ Parathyroidectomy ✕

Submit answer

Reference ranges

Score: **56.8%**

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Creatinine	71 µmol/l
Corrected calcium	2.71 mmol/l
Parathyroid hormone	10.2 pmol/l (1.0-7.0 pmol/l)

How should he be further managed?

Annual monitoring of calcium and renal function	7%
Encourage oral fluids	8%
Bisphosphonates	11%
Vitamin D supplementation	3%
Parathyroidectomy	71%

The correct answer is parathyroidectomy. This is a patient who has developed renal colic secondary to likely primary hyperparathyroidism, as is suggested by his hypercalcaemia and elevated parathyroid hormone. The mainstay of management of primary hyperparathyroidism is parathyroidectomy, but cases have to be appropriately identified as surgical candidates. This patient developed renal stones as a likely complication and therefore would benefit from surgery. If the blood tests been an incidental finding, then monitoring and oral fluids both would have been more appropriate.

Source:

'Hypercalcaemia.' Clinical Knowledge Summaries. National Institute for Health and Care Excellence, Dec. 2014.

👍 👎 💬 Discuss (3) Improve

Next question

Primary hyperparathyroidism ★

Primary hyperparathyroidism is caused by excess secretion of PTH resulting in hypercalcaemia. It is the most common cause of hypercalcaemia in outpatients and is often diagnosed following an incidental finding of an elevated serum calcium concentration. In 85% of cases a parathyroid adenoma is responsible.

Causes of primary hyperparathyroidism
- 85%: solitary adenoma
- 10%: hyperplasia
- 4%: multiple adenoma
- 1%: carcinoma

Around 80% of patients are asymptomatic and are diagnosed on routine blood tests. The symptomatic features of primary hyperparathyroidism may be remembered by the mnemonic: 'bones, stones, abdominal groans and psychic moans':
- polydipsia, polyuria
- depression

- anorexia, nausea, constipation
- peptic ulceration
- pancreatitis
- bone pain/fracture
- renal stones
- hypertension

Associations

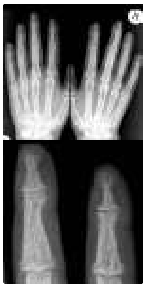
- hypertension
- multiple endocrine neoplasia: MEN I and II

Investigations

- bloods
  - raised calcium, low phosphate
  - PTH may be raised or (inappropriately, given the raised calcium) normal
- technetium-MIBI subtraction scan
- x-ray findings
  - pepperpot skull
  - osteitis fibrosa cystica

Treatment

- the definitive management is total parathyroidectomy
- conservative management may be offered if the calcium level is less than 0.25 mmol/L above the upper limit of normal AND the patient is > 50 years AND there is no evidence of end-organ damage
- patients not suitable for surgery may be treated with cinacalcet, a calcimimetic
  - a calcimimetic 'mimics' the action of calcium on tissues by allosteric activation of the calcium-sensing receptor



Next question

Textbooks

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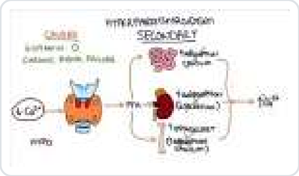
Extended textbook

Media


HYPERPARATHYROIDISM
SECONDARY

Understanding Hyperparathyroidism

Zero to Finals - YouTube 13 0

ENDOCRINOLOGY
HYPERPARATHYROIDISM

Hyperparathyroidism and the different types, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 2 0

Parathyroid Glands and Hyperparathyroidism

Norman Parathyroid Center - YouTube 👍 2 👎 1

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Question 90 of 217

An 18-year-old woman is brought into the hospital at midnight with generalised weakness and mild difficulty breathing.

On examination, there is weakness in the upper and lower limbs with proximal predominance. Reflexes in the upper limbs are reduced. Muscle tone is normal. Chest sounds clear but expansion is reduced bilaterally. GCS is 15/15.

The patient describes similar episodes in the past that have lasted for only minutes, the episodes decreased in frequency after commencing a ketogenic diet. They had been attributed to hemiplegic migraines, though she had had bilateral symptoms.

From the following options, what investigation will be most helpful in confirming the likely diagnosis?

- ○ CT head ×
- ○ Electromyography ×
- ○ Lumbar puncture ×
- ○ MRI brain and spinal cord ×
- ○ Urea & electrolytes ×

Submit answer

Reference ranges

Score: **57.3%**

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An 18-year-old woman is brought into the hospital at midnight with generalised weakness and mild difficulty breathing.

On examination, there is weakness in the upper and lower limbs with proximal predominance. Reflexes in the upper limbs are reduced. Muscle tone is normal. Chest sounds clear but expansion is reduced bilaterally. GCS is 15/15.

The patient describes similar episodes in the past that have lasted for only minutes, the episodes decreased in frequency after commencing a ketogenic diet. They had been attributed to hemiplegic migraines, though she had had bilateral symptoms.

From the following options, what investigation will be most helpful in confirming the likely diagnosis?

CT head	2%
Electromyography	14%
Lumbar puncture	3%
MRI brain and spinal cord	11%
✓ Urea & electrolytes	70%

Episodes of weakness, hypokalaemia, symptoms triggered by carbohydrate meals → hypokalaemic periodic paralysis

Important for me Less important

The most likely diagnosis is a periodic paralysis of which hypokalaemic periodic paralysis is the most common. Attacks can be precipitated by high-carbohydrate meals which explains why the number of episodes reduced for this patient when she commenced on a low-carbohydrate ketogenic diet.

Differentials might include myasthenia gravis (though this is commonly associated with fatigable weakness rather than 'attacks'), metabolic myopathies (which involve weakness typically worsened by exercise), or in a first attack Guillain-Barre, transverse myelitis, of infective paralysis (tick paralysis, botulism) may be considered.

Urea and electrolytes is the correct answer. A low potassium level would make the diagnosis here clearer. Average potassium levels in hypokalaemic periodic paralysis are around 2.4mmol/L. If hypokalaemia is significantly low, less than 2.0mmol/L for example, you should think about secondary causes of hypokalaemia such as losses from the gut, and renal losses.

MRI brain and spinal cord is incorrect. This would be a reasonable investigation to investigate the possibility of multiple sclerosis (MS). Here, the pattern of weakness does not fit a particular cerebral or spinal territory which you would expect with MS.

Electromyography is incorrect. Although this would be able to demonstrate myopathy during an acute attack, it would not assist us in determining the specific underlying diagnosis as there are many causes of myopathy.

Lumbar puncture is incorrect. A lumbar puncture would be helpful in looking for inflammatory or infective causes of neurological symptoms, but would not be able to confirm a diagnosis of hypokalaemic periodic paralysis. Given the episodic nature of the attacks, transverse myelitis or infection, for example, would not explain the symptoms here.

CT head is incorrect. This patient's symptoms are recurrent and do not correspond to a particular cerebral territory. A CT head would be unhelpful in confirming the likely diagnosis.

👍 👎 💬 Discuss (4) Improve

Next question

Hypokalaemic periodic paralysis ★

Hypokalaemic periodic paralysis is a rare autosomal dominant disorder characterised by episodes of paralysis, typically occur at night. The underlying defect is a mutation in muscle voltage-gated calcium channels. Attacks may be precipitated by carbohydrate meals

Management
- lifelong potassium supplementation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journall 👍 1 👎 1

Review of neurological channelopathies

Suggest link

Report broken link

Score: **57.8%**

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A 75-year-old man is admitted onto the ward with confusion. He has a past medical history of an ischaemic stroke 3 months ago. His drug history includes atorvastatin, ramipril and clopidogrel. He lives alone and has no package of care. On examination he has very poor mobility. His heart rate is 105 bpm and blood pressure 105/75 mmHg. His JVP is not visible.

Blood results are as follows:

Hb	138 g/l	Na^+	155 mmol/l
Platelets	$340 * 10^9$/l	K^+	4.9 mmol/l
WBC	$12.2 * 10^9$/l	Urea	12.6 mmol/l (6.2 the previous month)
Neuts	$8.4 * 10^9$/l	Creatinine	115 µmol/l (58 the previous month)

What is the most likely cause of the hypernatraemia?

- ○ Osmotic diuresis ✕
- ○ Diabetes insipidus ✕
- ○ Reduced water intake ✕
- ○ Hyperaldosteronism ✕
- ○ SIADH ✕

Submit answer

Reference ranges

Score: **57.8%**

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WBC	$12.2 * 10^9/l$	Urea	12.6 mmol/l (6.2 the previous month)
Neuts	$8.4 * 10^9/l$	Creatinine	115 µmol/l (58 the previous month)

What is the most likely cause of the hypernatraemia?

Osmotic diuresis	5%
Diabetes insipidus	17%
🟩 Reduced water intake	72%
Hyperaldosteronism	2%
🟥 SIADH	4%

Hypernatraemia associated with hypovolaemia occurs due to a free water deficit. Common causes include reduced water intake (e.g. elderly), GI losses (e.g. vomiting and diarrhoea), skin losses (e.g. burns), and renal losses (e.g. osmotic diuresis)

Important for me Less important

The patient is clinically and biochemically dehydrated. We are therefore dealing with a case of hypovolaemic hypernatraemia.

Hypernatraemia occurs most commonly due to

- Insufficient water (primarily in patients who either do not experience thirst normally, or cannot act on it).
- Hypotonic fluid loss (e.g. water loss in excess of salt loss) via either the skin, gut or kidneys
- Salt overload e.g. acute salt poisoning (hypertonic saline, hypertonic sodium bicarbonate), hyperaldosteronism

In an elderly person, with decreased mobility, the most likely explanation would be due to reduced water intake.

👍 👎 Discuss (4) Improve

Next question

Hypernatraemia ★

Causes of hypernatraemia

- dehydration
- osmotic diuresis e.g. hyperosmolar non-ketotic diabetic coma
- diabetes insipidus
- excess IV saline

Hypernatraemia should be corrected with great caution. Although brain tissue can lose sodium and potassium rapidly, lowering of other osmolytes (and importantly water) occurs at a slower rate, predisposing to cerebral oedema, resulting in seizures, coma and death1. Although there are no clinical guidelines by NICE or Royal College of Physicians at present, it is generally accepted that a rate of no greater than 0.5 mmol/hour correction is appropriate[1].

1. Reynolds RM, Padfield PL, Seckl JR; Disorders of sodium balance. BMJ. 2006 Mar 25;332(7543):702-5.

＋ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **57.1%**

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Question 92 of 217

A 66-year-old man presents to his general practitioner with increasing lethargy and confusion. Past medical history includes depression, Barrett's oesophagus, and benign prostatic hyperplasia. Current medications are sertraline 100mg, lansoprazole 30mg BD, and tamsulosin 400mg. Blood tests are as follows:

Hb	104 g/l	Na^+	118 mmol/l
Platelets	168* 10^9/l	K^+	4.1 mmol/l
WBC	8.7* 10^9/l	Urea	7.9mmol/l
Neuts	2.5* 10^9/l	Creatinine	173 µmol/l
Lymphs	3.0* 10^9/l	Eosin	0.6 * 10^9/l
Corrected calcium	3.01mmol/l		
Total protein	95g/l	Albumin	30g/l

Urinary sodium 7mmol/l
Urinary osmolality 100mOsm/kg
Plasma osmolality 280mOsm/kg

What is the most likely cause of this patient's hyponatraemia?

- ○ Syndrome of inappropriate ADH secretion due to underlying malignancy ×
- ○ Multiple myeloma ×
- ○ Psychogenic polydipsia ×
- ○ Adrenal insufficiency ×
- ○ Drug-induced syndrome of inappropriate ADH secretion ×

Submit answer

Reference ranges

Score: **57.1%**

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A 66-year-old man presents to his general practitioner with increasing lethargy and confusion. Past medical history includes depression, Barrett's oesophagus, and benign prostatic hyperplasia. Current medications are sertraline 100mg, lansoprazole 30mg BD, and tamsulosin 400mg. Blood tests are as follows:

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Platelets	168* 10^9/l	K^+	4.1 mmol/l
WBC	8.7* 10^9/l	Urea	7.9mmol/l
Neuts	2.5* 10^9/l	Creatinine	173 µmol/l
Lymphs	3.0* 10^9/l	Eosin	0.6 * 10^9/l
Corrected calcium	3.01mmol/l		
Total protein	95g/l	Albumin	30g/l

Urinary sodium 7mmol/l
Urinary osmolality 100mOsm/kg
Plasma osmolality 280mOsm/kg

What is the most likely cause of this patient's hyponatraemia?

🟥	Syndrome of inappropriate ADH secretion due to underlying malignancy	17%
🟩	Multiple myeloma	41%
	Psychogenic polydipsia	22%
	Adrenal insufficiency	3%
	Drug-induced syndrome of inappropriate ADH secretion	17%

Hyperlipidaemia can cause pseudohyponatraemia

Important for me Less important

This patient has pseudohyponatraemia secondary to raised protein levels from an underlying myeloma. Raised protein levels and hyperlipidaemia are both important causes to consider when investigating hyponatraemia. These patients have a normal total sodium but concentrations are skewed due to the high volume of lipids or protein.

Syndrome of inappropriate ADH secretion causes a euvolemic hyponatraemia. Patients have a urinary sodium >20mmol/l with raised urinary osmolality and normal/low plasma osmolality. Psychogenic polydipsia is rare and both urinary and plasma osmolalities are low in these patients. Adrenal insufficiency can cause hyponatraemia but the presence of hypercalcaemia, renal impairment, and raised total in this patient make myeloma the most likely underlying cause.

👍 👎 Discuss (10) Improve

Next question

Hyponatraemia ★

Hyponatraemia may be caused by water excess or sodium depletion. Causes of pseudohyponatraemia include hyperlipidaemia (increase in serum volume) or a taking blood from a drip arm. Urinary sodium and osmolarity levels aid making a diagnosis

Urinary sodium > 20 mmol/l

Sodium depletion, renal loss (patient often hypovolaemic)

- diuretics: thiazides, loop diuretics
- Addison's disease
- diuretic stage of renal failure

Patient often euvolaemic

- SIADH (urine osmolality > 500 mmol/kg)
- hypothyroidism

Urinary sodium < 20 mmol/l

Sodium depletion, extra-renal loss

- diarrhoea, vomiting, sweating
- burns, adenoma of rectum

Water excess (patient often hypervolaemic and oedematous)

- secondary hyperaldosteronism: heart failure, liver cirrhosis
- nephrotic syndrome
- IV dextrose
- psychogenic polydipsia

AI + Q 123

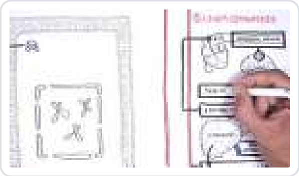
Next question

Textbooks

High-yield textbook

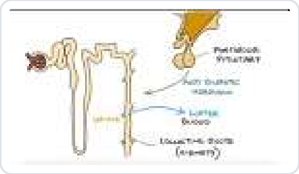
Extended textbook

Media



Hyponatraemia - classification, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 5 👎 0



Understanding Hyponatraemia

Zero To Finals - YouTube 👍 17 👎 1

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **56.5%**

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A 65-year-old retired nurse was referred to the endocrine clinic by her primary care physician in view of incidentally-detected hypercalcaemia. She had no significant past medical history and was not taking any regular medications. Her general physical and systemic examination was unremarkable.

Urea	7.5 mg/dl
Calcium	2.8 mmol/l
Phosphate	0.74mmol/l
Creatinine	98µmol/l
Alkaline phosphatase	450IU/l

Which one of the following investigations is most likely to help establish the diagnosis?

- ○ 1,25-OH vitamin D levels ×
- ○ PTH level ×
- ○ 25-OH vitamin D levels ×
- ○ Myeloma screen ×
- ○ Sestamibi scan ×

Submit answer

Reference ranges

Score: **56.5%**

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Calcium	2.8 mmol/l
Phosphate	0.74mmol/l
Creatinine	98μmol/l
Alkaline phosphatase	450IU/l

Which one of the following investigations is most likely to help establish the diagnosis?

1,25-OH vitamin D levels	6%
[✓ PTH level]	82%
25-OH vitamin D levels	2%
Myeloma screen	6%
Sestamibi scan	4%

Measuring PTH level is the first step in investigating hypercalcaemia

Important for me Less important

The most likely cause of hypercalcaemia in this case is primary hyperparathyroidism. The annual incidence is approximately 1 in 1000 and is commonly seen in post-menopausal women. The underlying aetiology of primary hyperparathyroidism is parathyroid adenoma (80-85%) followed by parathyroid hyperplasia (~10-14%). Measuring PTH level is the first step in investigating hypercalcaemia.

👍 👎 Discuss (4) Improve

Next question

Hypercalcaemia: causes ★

Two conditions account for 90% of cases of hypercalcaemia:

- 1. Primary hyperparathyroidism: commonest cause in non-hospitalised patients
- 2. Malignancy: the commonest cause in hospitalised patients. This may be due to a number of processes, including;
    - PTHrP from the tumour e.g. squamous cell lung cancer
    - bone metastases
    - myeloma,: due primarily to increased osteoclastic bone resorption caused by local cytokines (e.g. IL-1, tumour necrosis factor) released by the myeloma cells
- for this reason, measuring parathyroid hormone levels is the key investigation for patients with hypercalcaemia

Other causes include

- sarcoidosis
    - other causes of granulomas may lead to hypercalcaemia e.g. tuberculosis and histoplasmosis
- vitamin D intoxication
- acromegaly
- thyrotoxicosis
- Milk-alkali syndrome
- drugs:
    - thiazides
    - calcium-containing antacids
- dehydration
- Addison's disease
- Paget's disease of the bone

  - usually normal in this condition but hypercalcaemia may occur with prolonged immobilisation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians	👍 1 👎 1

Hypercalcaemia â€“ presentation and management

Suggest link | Report broken link

Media

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 4 👎 0

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

Report broken media

Score: **57%**

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Question 94 of 217

You review a 73-year-old female in the thyroid clinic. She has a history of hypothyroidism for about 25 years. Her other co-morbidities are COPD and dyslipidaemia. She is taking levothyroxine 125 mcg daily, atorvastatin 20mg daily, budesonide/formoterol inhaler 2 puffs BD and salbutamol inhaler PRN. Her most recent thyroid function tests showed:

TSH	0.4 mIU/l
FT4	29 pmol/l

You decide to reduce the dose of levothyroxine based on these results. What is the risk of over-replacement with levothyroxine?

- ○ Weight gain ×
- ○ Cognitive impairment ×
- ○ Liver failure ×
- ○ Osteoporosis ×
- ○ Agranulocytosis ×

Submit answer

Reference ranges

Score: **57%**

1 ×

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Question 94 of 217 ✓

You review a 73-year-old female in the thyroid clinic. She has a history of hypothyroidism for about 25 years. Her other co-morbidities are COPD and dyslipidaemia. She is taking levothyroxine 125 mcg daily, atorvastatin 20mg daily, budesonide/formoterol inhaler 2 puffs BD and salbutamol inhaler PRN. Her most recent thyroid function tests showed:

TSH	0.4 mIU/l
FT4	29 pmol/l

You decide to reduce the dose of levothyroxine based on these results. What is the risk of over-replacement with levothyroxine?

Weight gain	3%
Cognitive impairment	4%
Liver failure	4%
Osteoporosis	83%
Agranulocytosis	6%

Over-replacement with thyroxine increases the risk for osteoporosis

Important for me Less important

The major risks of over-replacement with thyroxine include osteoporosis and atrial fibrillation.

Weight gain is a feature of untreated hypothyroidism.

Cognitive impairment is not a recognised adverse effect of thyroxine.

Although levothyroxine can rarely cause mild elevations in liver enzymes, there have been no reports of liver failure.

Agranulocytosis is caused by anti-thyroid drugs.

👍 👎 Discuss Improve

Next question

Hypothyroidism: levothyroxine therapy ★

Key points

- initial starting dose of levothyroxine should be lower in elderly patients and those with ischaemic heart disease. The BNF recommends that for patients with cardiac disease, severe hypothyroidism or patients over 50 years the initial starting dose should be 25mcg od with dose slowly titrated. Other patients should be started on a dose of 50-100mcg od
- following a change in thyroxine dose thyroid function tests should be checked after 8-12 weeks
- the therapeutic goal is 'normalisation' of the thyroid stimulating hormone (TSH) level. As the majority of unaffected people have a TSH value 0.5-2.5 mU/l it is now thought preferable to aim for a TSH in this range
- women with established hypothyroidism who become pregnant should have their dose increased by at least 25-50 micrograms levothyroxine due to the increased demands of pregnancy. The TSH should be monitored carefully, aiming for a low-normal value
- there is no evidence to support combination therapy with levothyroxine and liothyronine

Side-effects of thyroxine therapy

- hyperthyroidism: due to over treatment
- reduced bone mineral density
- worsening of angina
- atrial fibrillation

Interactions

- iron, calcium carbonate
    - absorption of levothyroxine reduced, give at least 4 hours apart

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 15 👎 24

Management of hypothyroidism

Suggest link Report broken link

Media

HYPOTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hypothyroidism treatment medications

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **57.4%**

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Back to top

A 47-year-old man attends a diabetes follow-up appointment after being started on metformin 500mg TDS. His most recent HbA1c is 48mmol/mol and he has not been troubled by hypoglycaemia.

His past medical history includes atrial fibrillation and heart failure with preserved ejection fraction. He takes apixaban, bisoprolol, atorvastatin, and ramipril.

What would be the most appropriate course of action regarding his diabetic treatment?

- ○ Add in dapagliflozin ×
- ○ Add in glimepiride ×
- ○ Add in sitagliptin ×
- ○ No changes to current medications ×
- ○ Reduce metformin dose ×

Submit answer

Reference ranges

Score: **57.4%**

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A 47-year-old man attends a diabetes follow-up appointment after being started on metformin 500mg TDS. His most recent HbA1c is 48mmol/mol and he has not been troubled by hypoglycaemia.

His past medical history includes atrial fibrillation and heart failure with preserved ejection fraction. He takes apixaban, bisoprolol, atorvastatin, and ramipril.

What would be the most appropriate course of action regarding his diabetic treatment?

Add in dapagliflozin	69%
Add in glimepiride	0%
Add in sitagliptin	3%
No changes to current medications	26%
Reduce metformin dose	1%

In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

Important for me   Less important

The stem describes a patient with well-controlled type 2 diabetes mellitus and a background of heart failure. He is also on apixaban for atrial fibrillation and so at a higher risk of cardiovascular disease. All patients with type 2 diabetes and concomitant high risk of cardiovascular disease, chronic heart failure, or new cardiovascular disease, should be commenced on an SGLT-2 inhibitor once established on metformin if there are no contraindications.

Add in dapagliflozin is correct. Patients with a high risk of cardiovascular disease should be started on an SGLT-2 inhibitor such as dapagliflozin.

Add in glimepiride is incorrect. Glimepiride is a sulfonylurea, these drugs work by increasing insulin secretion and can be helpful for patients with symptomatic, recurrent hyperglycaemia. It can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Add in sitagliptin is incorrect. Sitagliptin is a DDP-4 inhibitor and can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Reduce metformin dose is incorrect, the current metformin dose is maintaining a good HbA1c and the patient is not troubled by hypo or hyperglycaemia. There would be no indication to reduce the dose in this case.

No changes to current medications would be appropriate if this patient had not developed atrial fibrillation. Due to the development of atrial fibrillation, his cardiovascular risk is much increased and as a result, the current best practice is to add in an SGLT-2 inhibitor such as dapagliflozin.

👍 👎 Discuss (2) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

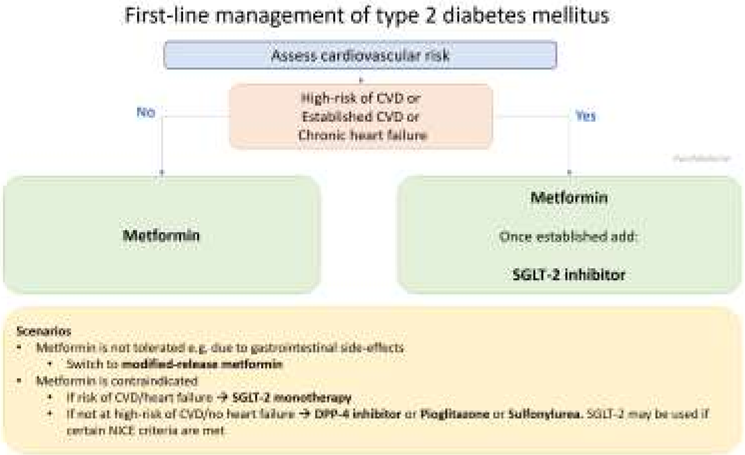
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios

- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

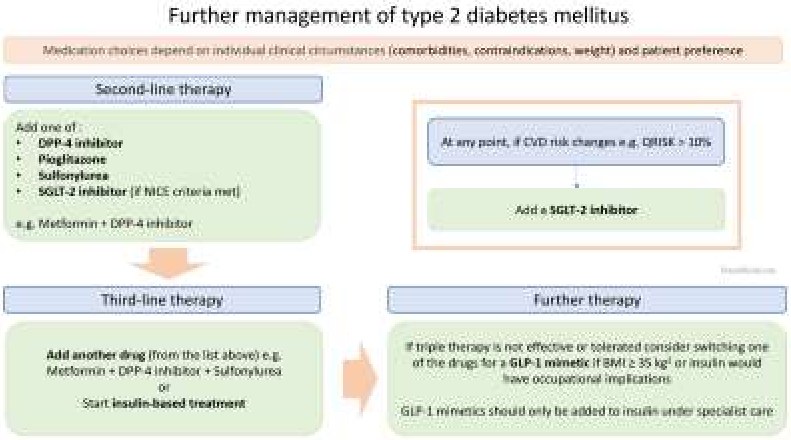
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

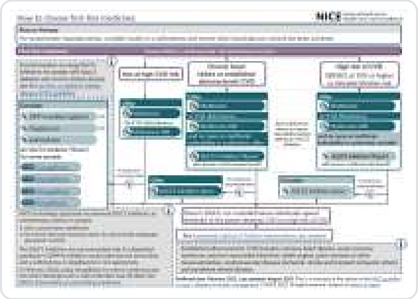
GLP-1 mimetics should only be added to insulin under specialist care

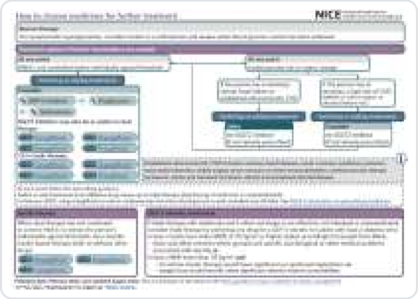
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

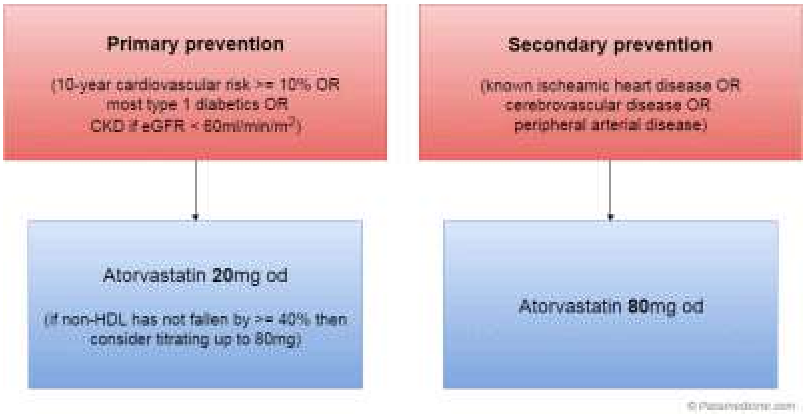
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

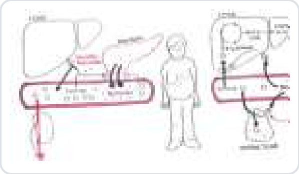
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

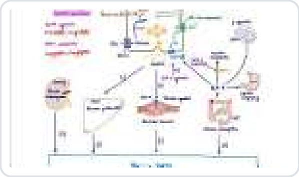
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **57.9%**

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A 39-year-old woman is referred to the outpatient department by her GP having been unsuccessfully treated with trimethoprim and nitrofurantoin for recurrent urinary tract infections. These have been occurring over the past six months. Over the last 2 days, the patient reports that urinating has become very painful and that the patient is now having difficulty urinating. The patient has a past medical history of type 2 diabetes mellitus. After completing a course of amoxicillin, the patient still complains of pain on urinating and pain in the lower abdomen. There is a family history of type 2 diabetes mellitus and the patient has a smoking history of 5 pack years, while drinking on average 15 units per week. An HbA1c test is performed and reveals a result of 82 mmol/mol.

What is the most likely organism causing the recurrent urinary tract infections?

- ○ Candida ×
- ○ Neisseria gonorrhoea ×
- ○ Chlamydia trachomatis ×
- ○ Trichomonas vaginalis ×
- ○ Mycoplasma genitalium ×

Submit answer

Reference ranges

Score: **57.9%**

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Question 96 of 217 ✓

A 39-year-old woman is referred to the outpatient department by her GP having been unsuccessfully treated with trimethoprim and nitrofurantoin for recurrent urinary tract infections. These have been occurring over the past six months. Over the last 2 days, the patient reports that urinating has become very painful and that the patient is now having difficulty urinating. The patient has a past medical history of type 2 diabetes mellitus. After completing a course of amoxicillin, the patient still complains of pain on urinating and pain in the lower abdomen. There is a family history of type 2 diabetes mellitus and the patient has a smoking history of 5 pack years, while drinking on average 15 units per week. An HbA1c test is performed and reveals a result of 82 mmol/mol.

What is the most likely organism causing the recurrent urinary tract infections?

Candida	70%
Neisseria gonorrhoea	3%
Chlamydia trachomatis	11%
Trichomonas vaginalis	4%
Mycoplasma genitalium	11%

The most likely organism in this scenario is candida - in those individuals who do have symptomatic Candida infections, the symptoms are indistinguishable from those caused by bacterial infections. Cystitis is associated with dysuria, urgency, suprapubic discomfort. This patients recent symptoms of oliguria, difficultly passing urine and painful urination suggest a complication such as the presence of a fungus ball.

Discuss (4) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice
- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015
- individual targets should be agreed with patients to encourage motivation

- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

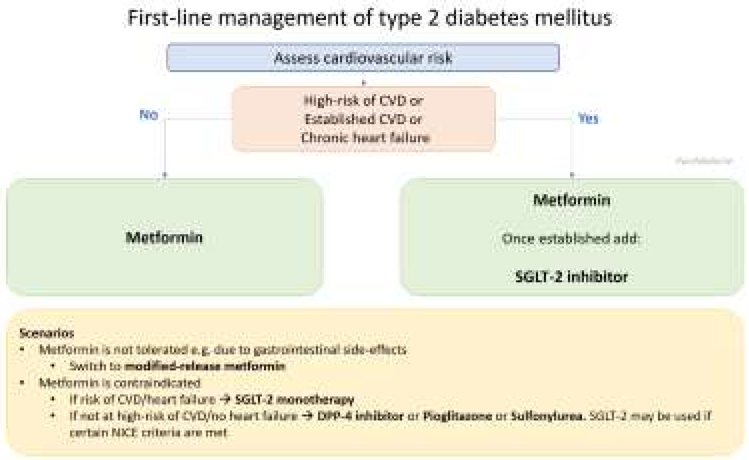
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

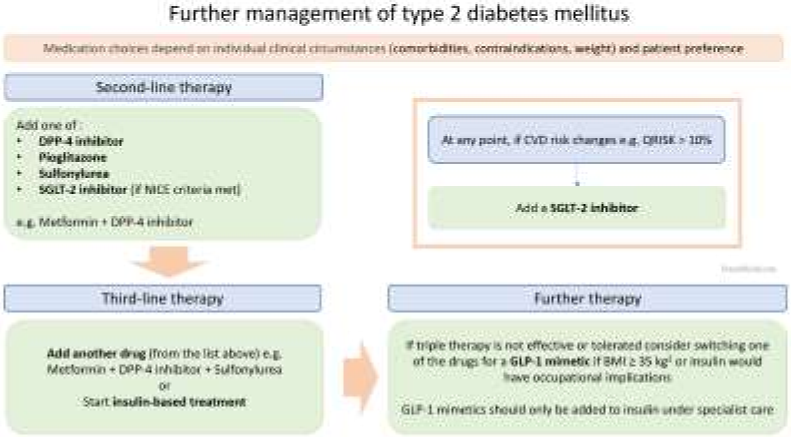
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:

    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of:
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea

- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

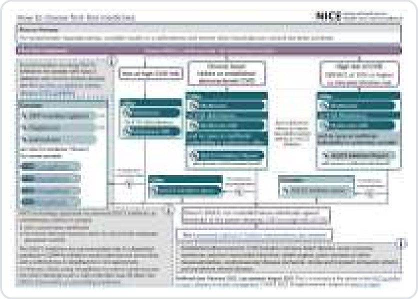
GLP-1 mimetics should only be added to insulin under specialist care

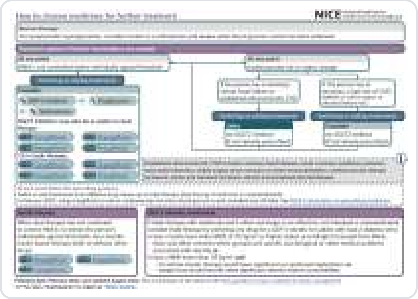
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line

    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

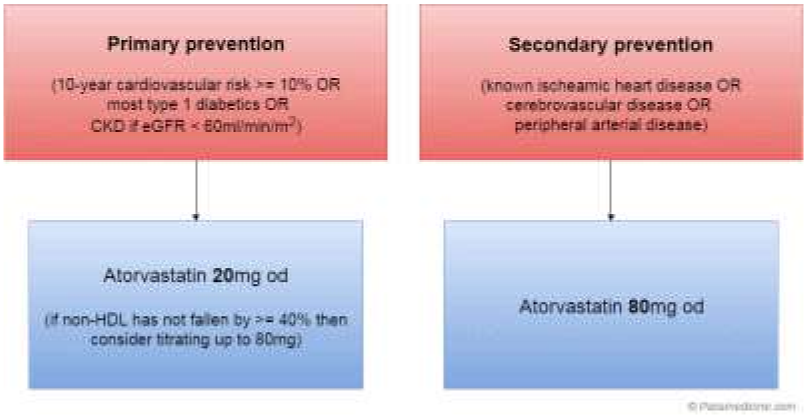
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischaemic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
© Passmedicine.com

Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

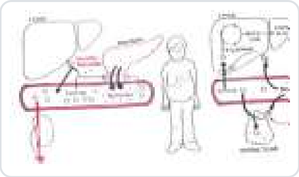
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

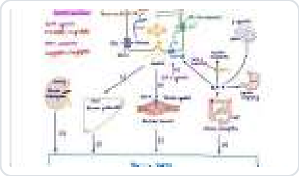
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **58.3%**

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A 58 year-old man presents with a two month history of weight loss and a one week history of increasing confusion. His partner reports that his clothes are now loose on him and that he has started to forget things and that he has been unable to reach for objects off the top shelf at the supermarket over the last two months due to increasing weakness. Six weeks ago he had been treated for an islet cell carcinoma of the pancreas with chemotherapy and has no other past medical history.

Examination reveals an abbreviated mental test score of 5/10 and weakness in the shoulders and getting out of the chair. Heart sounds 1 and 2 are present with no added sounds, his chest is clear and the abdomen is soft and non-tender.

Observations reveal a blood pressure of 158/95 mmHg, a pulse rate of 90 beats per minute, a temperature of 37.5ºC and a respiratory rate of 14 breaths per minute. Random blood glucose is 16.2 mmol/L.

Blood tests are performed and reveal:

Hb	14.2 g/l
Platelets	180 * 10^9/l
WBC	4.9 * 10^9/l
Na^+	150 mmol/l
K^+	2.6 mmol/l
Urea	5.2 mmol/l
Creatinine	100 µmol/l
Bilirubin	15 µmol/l
ALP	70 u/l
ALT	28 u/l
γGT	47 u/l
Albumin	48 g/l

What is the most likely diagnosis?

- ○ Paraneoplastic encephalitis ✕
- ○ Cerebral metastases ✕
- ○ Post chemotherapy Cushing's syndrome ✕
- ○ Post chemotherapy hypothyroidism ✕
- ○ Ectopic ACTH secretion ✕

Submit answer

Reference ranges ﹀

Score: **58.3%**

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Creatinine	100 µmol/l
Bilirubin	15 µmol/l
ALP	70 u/l
ALT	28 u/l
γGT	47 u/l
Albumin	48 g/l

What is the most likely diagnosis?

🟥	Paraneoplastic encephalitis	9%
	Cerebral metastases	5%
	Post chemotherapy Cushing's syndrome	35%
	Post chemotherapy hypothyroidism	3%
🟩	Ectopic ACTH secretion	48%

The confusion, hypertension and proximal myopathy, along with the hypernatraemia, hypokalaemia and hyperglycaemia all point towards a diagnosis of Cushing's syndrome. The subtype is most likely ectopic secretion of ACTH by the islet cell carcinoma, a neuroendocrine tumour and can release ectopic hormones. The post-chemotherapy Cushing's syndrome is unlikely, as the chemotherapy started after the proximal myopathy had begun to take effect. Further, in ectopic ACTH secretion, the hypokalaemia tends to be more pronounced, as in this case.

👍 👎 💬 Discuss (11) Improve

Next question

Cushing's syndrome: causes ★

It should be noted that exogenous causes of Cushing's syndrome (e.g. glucocorticoid therapy) are far more common than endogenous ones.

ACTH dependent causes

- Cushing's disease (80%): pituitary tumour secreting ACTH producing adrenal hyperplasia
- ectopic ACTH production (5-10%): e.g. small cell lung cancer is the most common causes

ACTH independent causes

- iatrogenic: steroids
- adrenal adenoma (5-10%)
- adrenal carcinoma (rare)
- Carney complex: syndrome including cardiac myxoma
- micronodular adrenal dysplasia (very rare)

Pseudo-Cushing's

- mimics Cushing's
- often due to alcohol excess or severe depression
- causes false positive dexamethasone suppression test or 24 hr urinary free cortisol
- insulin stress test may be used to differentiate

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ 👍 9 👎 13

Side-effects of systemic steroids

Suggest link Report broken link

Media

CUSHING SYNDROME

Cushing syndrome

Osmosis - YouTube 👍 1 👎 1

CUSHING'S SYNDROME

Understanding Cushing's Syndrome

Zero To Finals - YouTube 👍 0 👎 1

Report broken media

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Question 98 of 217

A 45-year-old man attends his general practitioner for an annual review of his diabetes for which he takes metformin. He had an NSTEMI three months and has been commenced on aspirin, clopidogrel, bisoprolol and ramipril.

Blood tests 6 months ago:

HbA1c	51 mmol/mol	(<48)

Blood tests today:

HbA1c	47 mmol/mol	(<48)

What is the correct management of type 2 diabetes in this patient at this point?

- ○ Add empagliflozin ×
- ○ Add gliclazide ×
- ○ Add pioglitazone ×
- ○ Add sitagliptin ×
- ○ No changes to current treatment ×

Submit answer

Reference ranges

Score: **57.7%**

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HbA1c	51 mmol/mol	(<48)

Blood tests today:

HbA1c	47 mmol/mol	(<48)

What is the correct management of type 2 diabetes in this patient at this point?

Add empagliflozin	69%
Add gliclazide	0%
Add pioglitazone	1%
Add sitagliptin	1%
No changes to current treatment	29%

In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

Important for me Less important

Add empagliflozin is correct. While this patient has well-controlled diabetes on metformin, SGLT-2 inhibitors should be introduced if at any point they developed cardiovascular disease (CVD). As he has had a recent NSTEMI, empagliflozin should be commenced.

Add gliclazide is incorrect. The patient has well-controlled diabetes and so the addition of gliclazide is not warranted.

Add pioglitazone is incorrect. The patient has well-controlled diabetes and so the addition of pioglitazone is not warranted. Additionally, you may be cautious about the possibility of heart failure in this patient.

Add sitagliptin is incorrect. The patient has well-controlled diabetes and so the addition of sitagliptin is not warranted.

No changes to current treatment is incorrect. While the HbA1c is well controlled, if at any point the patient develops CVD, an SGLT-2 inhibitor should be added.

👍 👎 Discuss (3) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice
- encourage high fibre, low glycaemic index sources of carbohydrates

- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

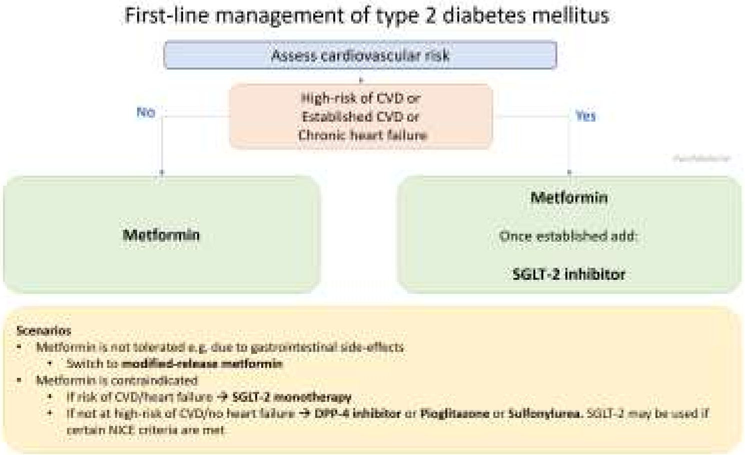
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

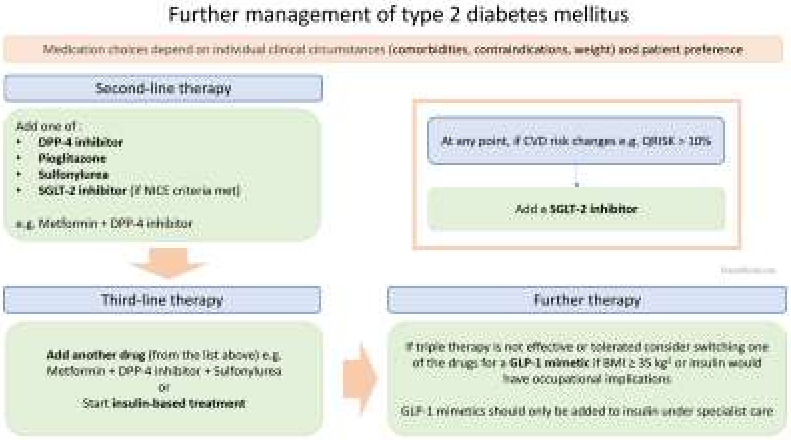
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met



Medication choices depend on individual clinical circumstances (**comorbidities, contraindications, weight**) and **patient preference**

Add one of :
- **DPP-4 inhibitor**
- **Pioglitazone**
- **Sulfonylurea**
- **SGLT-2 inhibitor** (if NICE criteria met)

e.g. Metformin + DPP-4 inhibitor

At any point, if CVD risk changes e.g. QRISK > 10%

Add a **SGLT-2 inhibitor**

Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start **insulin-based treatment**

If triple therapy is not effective or tolerated consider switching one of the drugs for a **GLP-1 mimetic** if BMI ≥ 35 kg¹ or insulin would have occupational implications

GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is

indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

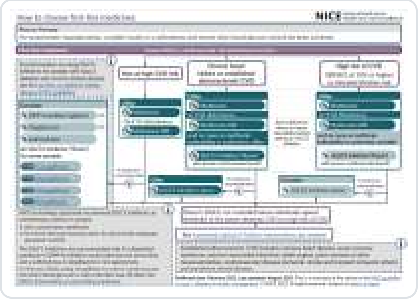
GLP-1 mimetics should only be added to insulin under specialist care

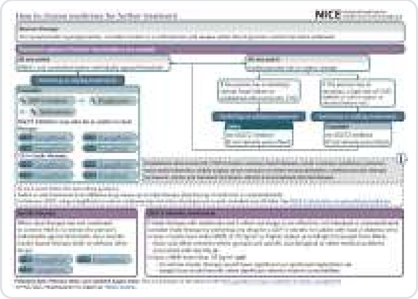
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

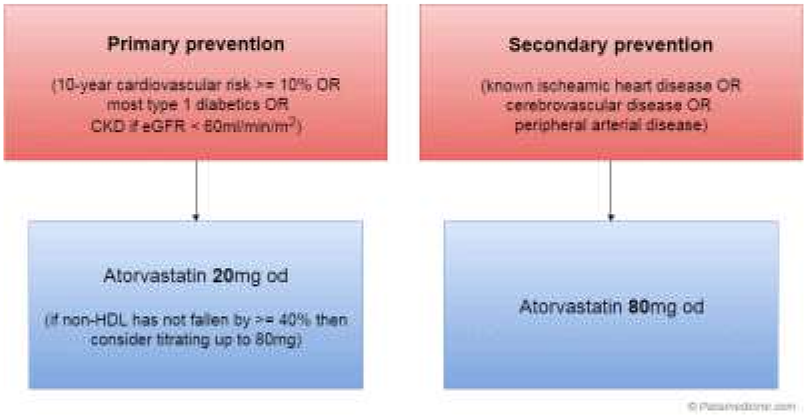
Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be

offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
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Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

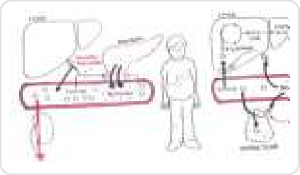
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

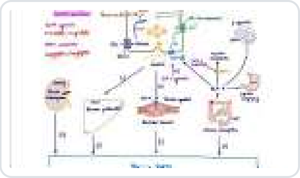
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

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Score: **58.2%**

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On examination, you notice she is flushed and tremulous but alert. She has no goitre and no rashes across her skin. There is a small mass palpable in the left side of the pelvis that is non tender and there are normal bowel sounds overlying. Her visual fields and cranial nerve exams are normal. A thyroid scintigraphy shows normal uptake. An MRI scan is awaited.

TSH	5.0 mU/L (range 0.4-4.0)
T4	28 pmol/l (range 9-24)
Urine HCG	negative

What is the most likely diagnosis?

- ○ Jod-Basedow phenomenon ×
- ○ Exogenous iodine intake ×
- ○ Pituitary adenoma ×
- ○ Molar pregnancy ×
- ○ Struma ovarii ×

Submit answer

Reference ranges

Score: **58.2%**

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Question 99 of 217 ✓

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TSH	5.0 mU/L (range 0.4-4.0)
T4	28 pmol/l (range 9-24)
Urine HCG	negative

What is the most likely diagnosis?

Jod-Basedow phenomenon	15%
Exogenous iodine intake	8%
Pituitary adenoma	11%
Molar pregnancy	7%
Struma ovarii	59%

Ovarian teratomas can produce exogenous TSH

Important for me Less important

This lady has clear secondary hyperthyroidism. There are no signs of pituitary disease so we have to consider other sources of TSH production. In pregnancy, HCG can act as an agonist at the TSH receptor but the test here is negative. The pelvic mass and TSH rise can be explained by ovarian teratomas that rarely can secrete TSH.

👍 👎 Discuss (11) Improve

Next question

Ovarian tumours

There are 4 main types of ovarian tumours

- surface derived tumours
- germ cell tumours
- sex cord-stromal tumours
- metastasis

Surface derived tumours

These account for around 65% of ovarian tumours, including the greatest number of malignant tumours.

Tumour	Benign/malignant	Notes
Serous cystadenoma	Benign	Most common benign ovarian tumour, often bilateral Cyst lined by ciliated cells (similar to Fallopian tube)
Serous cystadenocarcinoma	Malignant	Often bilateral Psammoma bodies seen (collection of calcium)

Tumour	Benign/malignant	Notes
Mucinous cystadenoma	Benign	Cyst lined by mucous-secreting epithelium (similar to endocervix)
Mucinous cystadenocarcinoma	Malignant	May be associated with pseudomyxoma peritonei (although mucinous tumour of appendix is the more common cause)
Brenner tumour	Benign	Contain Walthard cell rests (benign cluster of epithelial cells), similar to transitional cell epithelium. Typically have 'coffee bean' nuclei.

Germ cell tumours

These are more common in adolescent girls and are account for 15-20% of tumours. Similar cancer types to those seen in the testicle.

Tumour	Benign/malignant	Notes
Teratoma	Mature teratoma (dermoid cyst) - most common: benign Immature teratoma: malignant	Account for 90% of germ cell tumours Contain a combination of ectodermal (e.g. hair), mesodermal (e.g. bone) and endodermal tissue
Dysgerminoma	Malignant	Most common malignant germ cell tumour Histological appearance similar to that of testicular seminoma Associated with Turner's

Tumour	Benign/malignant	Notes
		syndrome Typically secrete hCG and LDH
Yolk sac tumour	Malignant	Typically secrete AFP Schiller-Duval bodies on histology are pathognomonic
Choriocarcinoma	Malignant	Rare tumour that is part of the spectrum gestational trophoblastic disease Typically have increased hCG levels Often characterised by early haematogenous spread to the lungs

Sex cord-stromal tumours

Represent around 3-5% of ovarian tumours. Often produce hormones.

Tumour	Benign/malignant	Notes
Granulosa cell tumour	Malignant	Produces oestrogen leading to precocious puberty if in children or endometrial hyperplasia in adults. Contains Call-Exner bodies (small eosinophilic fluid-filled spaces between granulosa cells)
Sertoli-Leydig cell tumour	Benign	Produces androgens → masculinizing effects Associated with Peutz-Jegher syndrome

Tumour	Benign/malignant	Notes
Fibroma	Benign	Associated with Meigs' syndrome (ascites, pleural effusion) Solid tumour consisting of bundles of spindle-shaped fibroblasts Typically occur around the menopause, classically causing a pulling sensation in the pelvis

Metastatic tumours

Account for around 5% of tumours.

Tumour	Benign/malignant	Notes
Krukenberg tumour	Malignant	Metastases from a gastrointestinal tumour resulting in a mucin-secreting signet-ring cell adenocarcinoma

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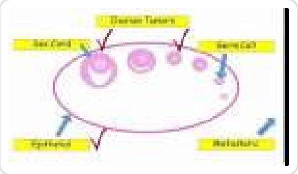
Next question

Textbooks

High-yield textbook

Extended textbook

Media


Ovarian Tumors
Sex Cord
Germ Cell
Epithelial
Metastatic

Ovarian tumors

12DaysinMarch - YouTube 👍 3 👎 0

Epithelial ovarian cancer

Osmosis - YouTube 👍 1 👎 0

SEX CORD-STROMAL OVARIAN CANCER

Sex cord-stromal ovarian cancer

Osmosis - YouTube 👍 1 👎 0

Germ cell ovarian cancer

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Score: **58.6%**

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Question 100 of 217

A 17-year-old girl is brought into the emergency department by her mother. The patient appears terrified after she experienced an episode on waking earlier in the morning when she could not move at all for 2 hours. This was her second episode. She reports no loss of consciousness and was aware throughout the episode. She has no other past medical history documented. She is not aware of a previous episode of epilepsy. On examination, her heart sounds and breath sounds are unremarkable. Neurological examination demonstrated no abnormalities. She has normal dentition and her body mass index is 19.5 kg/m². A 12 lead ECG demonstrated a jerky baseline with flat T waves. What is the most likely diagnosis?

- ○ Partial or absence seizures ✕
- ○ Guillain-Barre syndrome ✕
- ○ Botulinum toxicity ✕
- ○ Myasthenia gravis ✕
- ○ Hypokalaemia ✕

Submit answer

Reference ranges

Score: **58.6%**

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Question 100 of 217 ✓

A 17-year-old girl is brought into the emergency department by her mother. The patient appears terrified after she experienced an episode on waking earlier in the morning when she could not move at all for 2 hours. This was her second episode. She reports no loss of consciousness and was aware throughout the episode. She has no other past medical history documented. She is not aware of a previous episode of epilepsy. On examination, her heart sounds and breath sounds are unremarkable. Neurological examination demonstrated no abnormalities. She has normal dentition and her body mass index is 19.5 kg/m². A 12 lead ECG demonstrated a jerky baseline with flat T waves. What is the most likely diagnosis?

Partial or absence seizures	8%
Guillain-Barre syndrome	0%
Botulinum toxicity	2%
Myasthenia gravis	1%
[✓ Hypokalaemia]	89%

The patient describes episodes of periodic paralysis and the ECG characteristics are consistent with that of hypokalaemia. The underlying diagnosis is a rare familial condition of skeletal muscle ion channels called hypokalaemic periodic paralysis, which tends to develop in childhood and adolescence. Attacks last hours and the neurological examination is usually unremarkable in between attacks. The average potassium on diagnosis is 2.4 mmol/L[1]. Diagnosis is often made clinically in association with low potassium but genetic testing can help if known mutations are present.

1. Miller TM, Dias da Silva MR, Miller HA et al. Correlating phenotype and

genotype in the periodic paralyses. Neurology. 2004;63(9):1647.

Discuss (8) Improve

Next question

Hypokalaemic periodic paralysis ★

Hypokalaemic periodic paralysis is a rare autosomal dominant disorder characterised by episodes of paralysis, typically occur at night. The underlying defect is a mutation in muscle voltage-gated calcium channels. Attacks may be precipitated by carbohydrate meals

Management

- lifelong potassium supplementation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journall 👍 1 👎 1

Review of neurological channelopathies

Suggest link Report broken link

Score: **59%**

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Question 101 of 217

A 34-year-old woman is referred to endocrinology clinic for assessment after reporting symptoms of heat intolerance, tremors and diarrhoea to her General Practitioner. Blood tests in primary care showed evidence of thyrotoxicosis. Further assessment at clinic revealed the symptoms had been present for approximately 4 weeks. The patient had initially attributed the symptoms to the stress of caring for her new baby, who had been born 6 weeks previously. She denied any symptoms of pain on eye movements, diplopia or skin rashes.

Past medical history included only her recent pregnancy with delivery by vaginal delivery. The patient took no regular medications. There was no family history of thyroid disorders. Prior to taking maternity leave, the patient worked as a lawyer. She did not drink or smoke.

Examination revealed a small, diffuse and mildly tender goitre with no evidence of thyroid bruit. There was fine tremor of outstretched hands but no exophthalmos or proptosis. Investigations requested following clinic review are listed below.

Thyroid stimulating hormone	0.1 microU / L (reference 0.4-5.0)
T4 free serum	19.5 pmol / L (reference 8.5-15.2)
T3 free serum	8.1 pmol / L (reference 3.5-6.5)
Thyroid peroxidase antibodies	250 mU / L (reference < 150)
Erythrocyte sedimentation rate	21 ml / h

Thyroid scintiscanning (Technitium-99): no significant thyroid uptake

What is the most likely diagnosis?

○ Graves' disease ✕

○ Toxic thyroid nodule ✕

○ Viral thyroiditis ✕

○ Toxic multinodular goitre ✕

○ Post-partum thyroiditis ✕

Submit answer

Reference ranges ∨

Score: **59%**

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Thyroid peroxidase antibodies	250 mU / L (reference < 150)
Erythrocyte sedimentation rate	21 ml / h

Thyroid scintiscanning (Technitium-99): no significant thyroid uptake

What is the most likely diagnosis?

Graves' disease	10%
Toxic thyroid nodule	1%

Viral thyroiditis	12%
Toxic multinodular goitre	1%
Post-partum thyroiditis	75%

Post-partum thyroiditis is associated with elevated thyroid peroxidase antibodies but no significant uptake on thyroid scintiscanning and is, therefore, the correct answer in this case given that symptoms began approximately two weeks postpartum.

Viral thyroiditis is usually associated with an elevated ESR and negative anti-thyroid antibodies. Graves' disease, toxic multinodular goitre and a toxic thyroid nodule would be associated with different patterns of uptake on thyroid scintiscanning.

Weetman A. Investigating low thyroid stimulating hormone (TSH) level. BMJ 2013;347:f6842.

👍 👎 Discuss (12) Improve

Next question

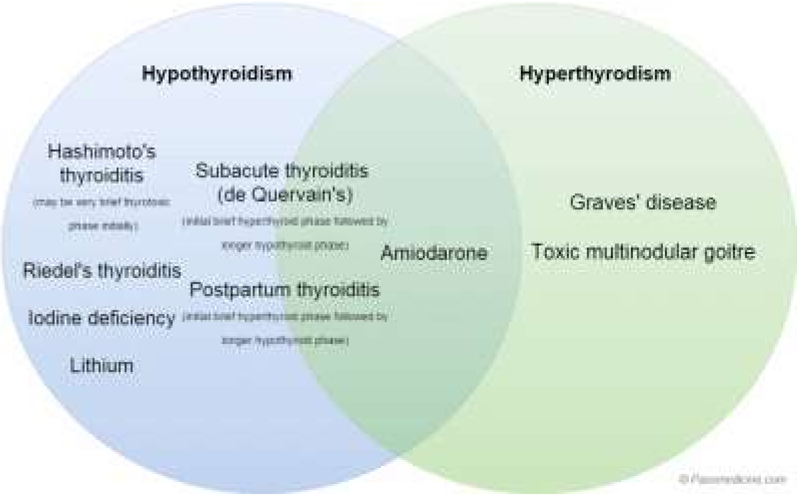
Postpartum thyroiditis

Three stages
- 1. Thyrotoxicosis
- 2. Hypothyroidism
- 3. Normal thyroid function (but high recurrence rate in future pregnancies)

Thyroid peroxidase antibodies are found in 90% of patients

Management
- thyrotoxic phase

    - propranolol is typically used for symptom control
    - not usually treated with anti-thyroid drugs as the thyroid is not overactive.
- hypothyroid phase
    - usually treated with thyroxine


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

POSTPARTUM
THYROIDITIS

Postpartum thyroiditis

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **59.4%**

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A 25-year-old woman is brought to the emergency department by ambulance after being found unwell by friends. Collateral history reported by the paramedics indicated that the patient had been unwell for 3 days with vomiting and diarrhoea. Her housemate said that the patient had been unable to eat since becoming unwell and that he did not think she had been taking her regular insulin during that time. The patient herself was too disorientated to give any history. The paramedics had found both novorapid and lantus insulin pen devices in the patients fridge.

General examination indicated a drowsy and dehydrated patient with generalised abdominal tenderness but no evidence of focal peritonism.

Please see below for selected investigation results.

Observations: blood pressure 86 / 57 mmHg; heart rate 127 beats per minute; respiratory rate 28 per minute; O2 saturations 100 % (room air); Temperature 37.1 oC.

Fingerpick blood glucose	38.2 mmol / L
Fingerpick blood ketones	8.7 mmol / L
Urea	12.5 mmol / L
Creatinine	123 micromol / L
Sodium	148 mmol / L
Potassium	3.7 mmol / L
Haemoglobin	156 g / dL
White cell count	14.3 x 10>3 / microlitre
Neutrophils	11.3 x 10>3 / microlitre
Platelets	453 x 10>3 / microlitre

Arterial blood gas (room air)

pH	7.05

PaCO2	15 mmHg (reference 32-43)
PaO2	99 mmHg (reference 70-100)
Bicarbonate	12.3 mmol / L (reference 20.0-26.0)
Chloride	111 mmol / L (reference 99-108)
Lactate	7.5 mmol / L

What is the appropriate strategy for intravenous insulin treatment in this patient?

○ Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once acidosis resolved ×

○ Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally ×

○ Fixed rate insulin infusion following initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally ×

○ Variable rate insulin infusion with initial insulin bolus, converting to subcutaneous insulin once acidosis resolved ×

○ Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once ketonaemia resolved ×

Submit answer

Reference ranges ﹀

Score: **59.4%**

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Chloride	111 mmol / L (reference 99-108)
Lactate	7.5 mmol / L

What is the appropriate strategy for intravenous insulin treatment in this patient?

	Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once acidosis resolved	3%
🟩	Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally	59%
🟥	Fixed rate insulin infusion following initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally	30%
	Variable rate insulin infusion with initial insulin bolus, converting to subcutaneous insulin once acidosis resolved	5%
	Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once ketonaemia resolved	3%

The patient is presenting in diabetic ketoacidosis due to vomiting, dehydration and omission of prescribed insulin.

The Joint British Diabetes Society recommend an insulin infusion at rate 0.1 units / kg / h. An initial bolus of insulin is not advised due to a randomised controlled trial that found no benefit. Fixed rate insulin infusions are now preferred over titration of insulin dose against blood sugar levels (sliding scale). This is due to the fact that blood glucose may correct more quickly than ketoacidosis and so ensures adequate insulin to eradicate ketones.

There is no consensus between expert bodies as to biochemical end-point of DKA, therefore it is advised that patients are transferred onto subcutaneous insulin once they are eating and drinking normally. It is vital to ensure an overlap between the administration of intravenous and subcutaneous insulin to avoid recurrent ketogenesis.

Misra S, Oliver N. Diabetic ketoacidosis in adults. BMJ 2015;351:h5660.

👍 👎 Discuss (3) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	Joint British Diabetes Societies (2013)
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
    - most patients with DKA are deplete around 5-8 litres
    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:

- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

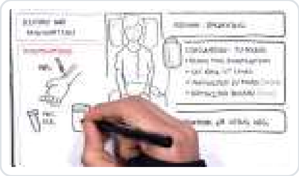
Joint British Inpatient Diabetes Societies Inpatient Care Group 👍 33 👎 23

The Management of Diabetic ketoacidosis in adults

Suggest link

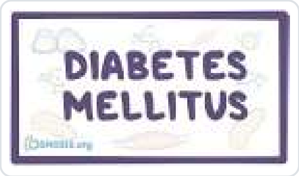
Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **58.8%**

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A 32 year-old man is referred by his GP after collapsing while at work. He does not remember the episode but witnesses say that there was no incontinence or fitting and the patient does not have a sore mouth or tongue. This is the first time this has happened and the patient does not have any other past medical history of note and takes no regular medication.

Examination reveals a blood pressure of 162/95 mmHg, a pulse of 74 beats per minute, a respiratory rate of 16 and a temperature of 37.4ºC. Heart sounds 1 and 2 are present with no added sounds, the lung fields are clear and his abdomen is soft and non-tender.

Blood tests performed and reveal:

Na^+	143 mmol/l
K^+	3.0 mmol/l
Urea	5.6 mmol/l
Creatinine	76 µmol/l
Bicarbonate	31 mmol/l
Renin	low
Aldosterone	low

Which of the following is the best treatment?

○ Amiloride ×

○ Bumetanide ×

○ Spironolactone ×

○ ACE inhibitor ×

○ Angiotensin II receptor blocker ✕

Submit answer

Reference ranges

Score: **58.8%**

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Creatinine	76 µmol/l
Bicarbonate	31 mmol/l
Renin	low
Aldosterone	low

Which of the following is the best treatment?

Amiloride	66%
Bumetanide	2%
Spironolactone	27%
ACE inhibitor	4%

Angiotensin II receptor blocker 1%

This man has Liddle's syndrome, an autosomal dominant disorder characterised by hypertension associated with hypokalaemic metabolic alkalosis, low plasma renin activity, and suppressed aldosterone secretion. Amiloride is the best treatment for the hypertension and hypokalaemia as it acts on the sodium channels directly, as opposed to spironolactone, which acts on mineralocorticoid receptors.

Discuss (6) Improve

Next question

Liddle's syndrome

Liddle's syndrome is a rare autosomal dominant condition that causes hypertension and hypokalaemic alkalosis. It is thought to be caused by disordered sodium channels in the distal tubules leading to increased reabsorption of sodium.

Treatment is with either amiloride or triamterene

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Media

LIDDLE'S SYNDROME
The Medics Guide

Liddle's syndrome

The Medics Guide - YouTube 👍 16 👎 0

Report broken media

Score: **59.2%**

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A 78-year-old female attends the diabetes clinic. She has longstanding type 2 diabetes. Over the last few years she has become increasingly frail. Her main complaint is recurrent nausea and vomiting. Earlier this year she underwent endoscopy and gastric emptying studies which confirmed gastroparesis. She has since been started on metoclopramide which has had minimal effect on her symptoms. Her weight has decreased by 10% over the past year with a current BMI of 26 kg/m².

Her HbA1c today at the clinic is 44 IFCC mmol/l (6.2%) having been 60 IFCC mmol/l (7.6%) this time last year.

Her past medical history includes chronic kidney disease stage 3 and aortic stenosis.

Her current therapy is Humulin M3 22 units at breakfast and dinner, metformin 500mg BD, ramipril 5mg OD, bendroflumethiazide 2.5mg OD, aspirin 75 mg OD.

She lives alone and is still driving. She denies the need for carers, however, she has had 3 falls in the past month. She describes particular difficulty getting up in the morning and says her mood can often be low in the mornings.

She checks her blood sugar once daily in the morning with the following results.

Saturday	3.1 mmol/l
Sunday	4.0 mmol/l
Monday	4.1 mmol/l
Tuesday	3.2 mmol/l
Wednesday	14.6 mmol/l
Thursday	3.5 mmol/l
Friday	16.1 mmol/l

What is the correct step in the management of her diabetes?

- ○ Change Humulin M3 to a glucagon-like-peptide 1 receptor agonist ×
- ○ Stop metformin ×
- ○ Add sitagliptin ×
- ○ Change Humulin M3 to 20 units in the morning and 10 units in the evening ×
- ○ Change Humulin M3 to 30 units once daily ×

Submit answer

Reference ranges

Score: **59.2%**

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Wednesday	14.6 mmol/l
Thursday	3.5 mmol/l
Friday	16.1 mmol/l

What is the correct step in the management of her diabetes?

Change Humulin M3 to a glucagon-like-peptide 1 receptor agonist	12%
Stop metformin	9%
Add sitagliptin	3%
[✓ Change Humulin M3 to 20 units in the morning and 10 units in the evening]	69%
Change Humulin M3 to 30 units once daily	6%

This lady's fasting blood sugars are too low. The 2 high readings raise the concern of overnight hypoglycaemia with reflex hyperglycaemia in the morning. Her recent weight loss will mean her insulin resistance will have decreased (as reflected in her decreasing HbA1c) and she will require smaller doses of insulin. Changing her insulin from a total of 44 units daily to 30 units daily offers protection against hypoglycaemia. Furthermore altering her split of insulin to the conventional 2/3rd in the morning and 1/3rd in the evening reduces the risk of overnight hypos.

The risks of tight glycaemic control in this lady vastly outweigh any benefits. She should be advised to check her sugar more regularly for the short-term and given advice regarding driving. Close follow-up with the diabetic nurses should be arranged until her hypo risk is reduced.

There is no indication for a GLP1 agonist (BMI 26) and metformin should continue (assuming eGFR remains above 30) to allow the reduction in her insulin doses to lowest effective levels.

👍 👎 Discuss (5) Improve

Next question

Insulin therapy: side-effects

Hypoglycaemia

- patients should be taught the signs of hypoglycaemia: sweating, anxiety, blurred vision, confusion, aggression
- conscious patients should take 10-20g of a short-acting carbohydrate (e.g. a glass of Lucozade or non-diet drink, three or more glucose tablets, glucose gel)
- every person treated with insulin should have a glucagon kit for emergencies where the patient is not able to orally ingest a short-acting carbohydrate
- patients who have frequent hypoglycaemic episodes may develop reduced awareness. If this develops then allowing glycaemic control to slip for a period of time may restore their awareness
- beta-blockers reduce hypoglycaemic awareness

Lipodystrophy

- typically presents as atrophy/lumps of subcutaneous fat
- can be prevented by rotating the injection site
- may cause erractic insulin absorption

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **59.6%**

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Back to top

A 55-year-old female presents with light-headedness and abdominal pain. She has a past medical history of asthma for which she takes regular beclometasone and as required salbutamol. On examination her blood pressure is 95/75 mmHg and heart rate 115 beats per minute.

Blood results are as follows:

Hb	135 g/l	Na$^+$	129 mmol/l
Platelets	352 * 10^9/l	K$^+$	5.2 mmol/l
WBC	14.2 * 10^9/l	Urea	10.2 mmol/l
Neuts	10.3 * 10^9/l	Creatinine	115 µmol/l
Lymphs	2.2 * 10^9/l	CRP	8 mg/l

A short Synacthen test is performed:

Time (minutes)	0	30	60
Cortisol (nmol/l)	150	165	212

A long Synacthen test is then performed:

Time (hours)	1	2	8	24
Cortisol (nmol/l)	202	420	820	1626

What is the most likely cause?

○ Adrenal Cushing's syndrome ✕

○ Secondary adrenal insufficiency ✕

○ Primary adrenal insufficiency (Addison's disease) ✕

○ Cushing's disease ×

○ Iatrogenic adrenal insufficiency ×

Submit answer

Reference ranges

Score: **59.6%**

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Back to top

Question 105 of 217 ✓

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Time (hours)	1	2	8	24
Cortisol (nmol/l)	202	420	820	1626

What is the most likely cause?

Adrenal Cushing's syndrome	3%
Secondary adrenal insufficiency	50%
Primary adrenal insufficiency (Addison's disease)	22%

Cushing's disease	5%
Iatrogenic adrenal insufficiency	20%

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

Important for me Less important

The clinical and biochemical results are suggestive of adrenal insufficiency. Biochemical results which suggest adrenal insufficiency include hyponatraemia, hyperkalaemia, a normal anion gap metabolic acidosis and hypoglycaemia.

The short Synacthen test demonstrates failure of cortisol to rise which confirms the diagnosis of adrenal insufficiency. A normal response is defined as a 30 minute serum cortisol concentration greater than 420 nmol/L.

The next step is to localise the lesion. This can be achieved by measurement of ACTH or by performing the long Synacthen test. Interpretation of this test is governed by the following:

- 1. In primary adrenal failure: we would not expect there to be a significant rise in cortisol during the long Synacthen rest since the adrenal glands are intrinsically dysfunctional
- 2. In secondary adrenal failure: chronically low levels of ACTH due to pituitary failure result in atrophy of the adrenal glands. Prolonged stimulation of the adrenal glands by ACTH in the long Synacthen test results in a degree of recovery by the adrenal glands resulting in a significant rise in cortisol. A response that rises gradually to a peak at 24 hours occurs in secondary adrenal failure. This pattern of results also occurs due to prolonged corticosteroid therapy use.

An important point is to remember that in some cases of long-standing adrenal atrophy due to secondary adrenal insufficiency, the adrenal glands will not respond even after 24 hours and will require several daily doses of depot Synacthen before an adrenal response is seen. The majority of these cases should be identifiable by measurement of plasma ACTH, which would be expected to be very low (in contrast to primary adrenal insufficiency where ACTH levels are very high).

In this case, the long Synacthen test demonstrates a cortisol which rises gradually to a peak at 24 hours confirming the diagnosis of secondary adrenal failure. The differential is now between iatrogenic adrenal insufficiency and secondary adrenal insufficiency due to pituitary disease. The patient is on inhaled steroids for asthma, however this is highly unlikely to cause adrenal insufficiency unless the patient was on a liver enzyme inhibitor (e.g. ketoconazole). Therefore the most likely diagnosis is secondary adrenal insufficiency.

👍 👎 Discuss (13) Improve

Next question

Addison's disease: investigations ★

In a patient with suspected Addison's disease the definite investigation is an ACTH stimulation test (short Synacthen test). Plasma cortisol is measured before and 30 minutes after giving Synacthen 250ug IM. Adrenal autoantibodies such as anti-21-hydroxylase may also be demonstrated.

If an ACTH stimulation test is not readily available (e.g. in primary care) then sending a 9 am serum cortisol can be useful:

- > 500 nmol/l makes Addison's very unlikely
- < 100 nmol/l is definitely abnormal
- 100-500 nmol/l should prompt a ACTH stimulation test to be performed

Associated electrolyte abnormalities are seen in around one-third of undiagnosed patients:

- hyperkalaemia
- hyponatraemia
- hypoglycaemia
- metabolic acidosis

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

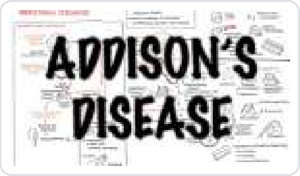
Links

Royal College of Physicians 👍 14 👎 7

2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media



Addison's disease

Armando Hasudungan - YouTube 👍 2 👎 1

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **60%**

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A 22-year-old gentleman presents to endocrinology clinic. He was referred by his GP as he was complaining of low energy and a morning testosterone was low. He immigrated from Albania one year ago and has been noticed to be always tired and lacking energy. He has also found to have low sexual desire after starting a new relationship in the UK. On examination he is tall and slim, with slight gynaecomastia. What is the most appropriate investigation after confirming the low morning testosterone?

- ○ Prolactin ✕
- ○ LH and FSH ✕
- ○ MRI pituitary ✕
- ○ Morning cortisol ✕
- ○ Synacthen test ✕

Submit answer

Reference ranges ⌄

Score: **60%**

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Question 106 of 217 ✓

A 22-year-old gentleman presents to endocrinology clinic. He was referred by his GP as he was complaining of low energy and a morning testosterone was low. He immigrated from Albania one year ago and has been noticed to be always tired and lacking energy. He has also found to have low sexual desire after starting a new relationship in the UK. On examination he is tall and slim, with slight gynaecomastia. What is the most appropriate investigation after confirming the low morning testosterone?

Prolactin	17%
LH and FSH	71%
MRI pituitary	7%
Morning cortisol	3%
Synacthen test	2%

A patient with low testosterone should have LH and FSH tested as first line investigation to separate primary and secondary hypogonadism

Important for me Less important

This is a patient with biochemical and clinical evidence of low testosterone. He is symptomatic with low energy and low sexual desire, and most likely would have erectile problems if questioned. The history of immigration is important as it raises the possibility of missed childhood diagnoses, in this case implying a missed chromosomal disorder. The most appropriate investigation to differentiate primary from secondary hypogonadism is LH and FSH. If LH and FSH are elevated this would suggest a primary hypogonadism such as Klinefelter's syndrome, as is suggested by the description of this patient, but if it is not then a secondary cause should be found. If that were the case, then MRI of pituitary can be necessary, as well as morning cortisol and prolactin. Synacthen testing should be done if morning cortisol is low to confirm adrenal insufficiency.

Discuss (5) Improve

Next question

Hypogonadism in men

Causes of hypogonadism in men
- primary (disease of the testes)
  - childhood mumps
- secondary (disease of the hypothalamus or pituitary)
  - Klinefelter syndrome
  - Kallman syndrome

Testosterone levels in men also tend to decline with age.

Testosterone deficiency causes a number of changes in men:
- loss of libido
- erectile dysfunction
- lethargy
- decreased muscle mass and strength
- reduced facial hair growth
- impaired glucose tolerance

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

KLINEFELTER SYNDROME

Klinefelter syndrome

Osmosis - YouTube 👍 6 👎 1

KALLMANN SYNDROME

Kallman's syndrome

Osmosis - YouTube 👍 5 👎 1

Report broken media

Score: **60.4%**

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Question 107 of 217 ✗

A 71-year-old gentleman presents to clinic for review. He has recently been diagnosed with type two diabetes following screening by his GP. His HbA1c has not responded to dietary changes that he was advised on. He has a past medical history of bladder cancer for which he had chemotherapy, hypertension, macular degeneration, eczema and chronic kidney disease. His baseline eGFR is 28ml/min/1.73m2.

Blood tests:

Na^+	139 mmol/l
K^+	4.4 mmol/l
Urea	6.2 mmol/l
Creatinine	214 μmol/l

What is the most appropriate medication to start?

	Metformin	12%
🟩	Sulfonylurea	40%
	Pioglitazone	4%
🟥	Insulin	27%
	GLP-1 mimetic	18%

The correct answer is a sulfonylurea. This is a patient with new type two diabetes and initial treatment would normally be metformin, but since the eGFR is <30ml/minute/1.73m2 metformin is contraindicated. The next step would be either a a dipeptidyl peptidase4 inhibitor, pioglitazone or a sulfonylurea. However, pioglitazone should not be offered to a patient with a history of bladder cancer. Therefore that leaves sulfonylurea as the correct answer. Insulin and GLP-1 mimetic could be used further down in the treatment algorithm.

Source:

'Type 2 diabetes in adults: management.' NICE guideline [NG28]. The National Institute for Health and Care Excellence, December 2015.

👍 👎 Discuss (7) Improve

Next question

Diabetes mellitus: management of type 2

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

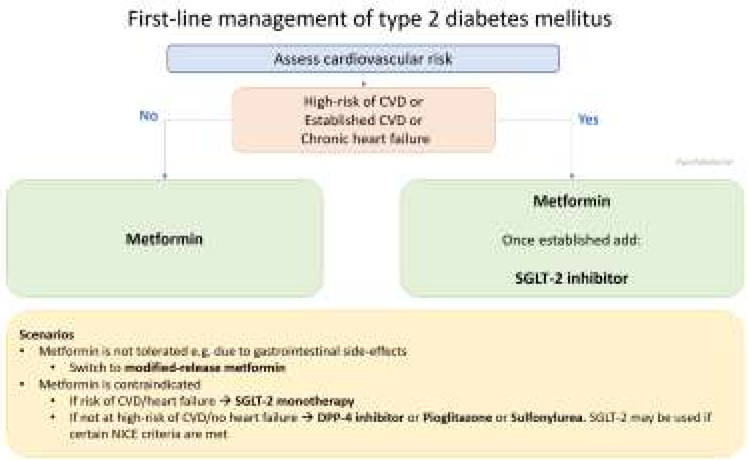
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

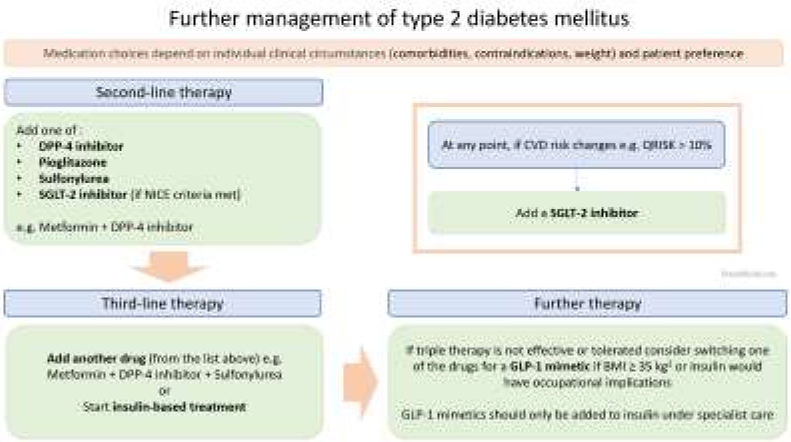
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a

  QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone

- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

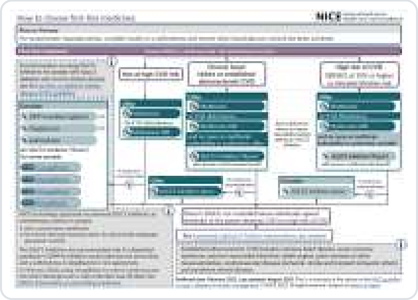
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

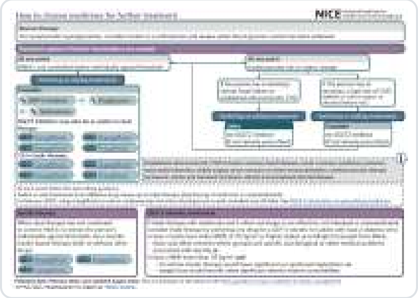
GLP-1 mimetics should only be added to insulin under specialist care

Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

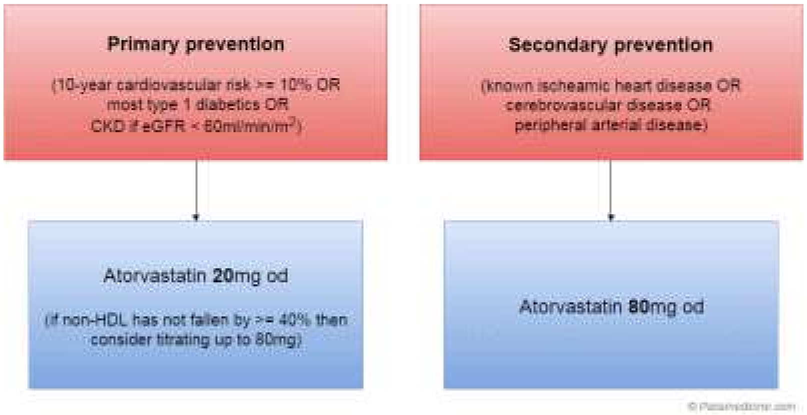
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2014 Lipid modification guidelines	33	40
SIGN 2010 Management of diabetes	36	41

NICE 👍 56 👎 48

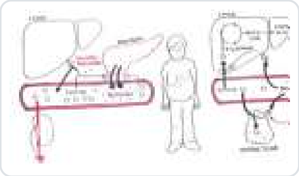
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

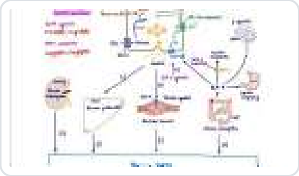
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **59.8%**

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107 ✗

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A 22-year-old female presents with nausea. She states that it has been going on for approximately 10 weeks. On systemic enquiry she also complains of constipation and fatigue. She has no past medical history and takes no regular medicines.

Blood results are as follows:

Hb	110 g/l	Na^+	142 mmol/l
Platelets	422 * 10^9/l	K^+	3.8 mmol/l
WBC	8.2 * 10^9/l	Urea	6.2 mmol/l
Neuts	5.8 * 10^9/l	Creatinine	52 µmol/l
Lymphs	2.8 * 10^9/l	CRP	4 mg/l

Thyroid function tests are also performed:

TSH	2.2 mIU/L (normal 0.4 - 4.0)
Total T3	3.9 nmol/l (normal 1.1-2.7)
Total T4	190 nmol/l (normal 60-160)
Free T3	3.6 pmol/l (normal 1.2 - 8.2)
Free T4	16 nmol/l (normal 11.7-31.2)

What investigation will you perform next?

- ○ Thyroid ultrasound ×
- ○ Thyroid peroxidase antibody levels ×
- ○ Thyroglobulin levels ×
- ○ Pituitary function tests ×

○ Pregnancy test ×

Submit answer

Reference ranges

Score: **59.8%**

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Back to top

Question 108 of 217 ✓

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Total T4	190 nmol/l (normal 60-160)
Free T3	3.6 pmol/l (normal 1.2 - 8.2)
Free T4	16 nmol/l (normal 11.7-31.2)

What investigation will you perform next?

Thyroid ultrasound	2%
Thyroid peroxidase antibody levels	7%
Thyroglobulin levels	8%
Pituitary function tests	2%

Pregnancy test 80%

Raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin, which can be seen during pregnancy

Important for me Less important

The patient has raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin. This pattern of results is commonly seen during pregnancy. Her symptoms could be attributed to hypothyroidism, however equally they are common symptoms in pregnancy. She is a female of child rearing age and therefore a pregnancy test should be performed.

Thyroid peroxidase antibody and thyroglobulin levels should be measures in suspected autoimmune hypothyroidism. The normal TSH, fT3 and fT4 makes autoimmune hypothyroidism very unlikely.

There is nothing biochemically or clinically to suggest pituitary disease.

A thyroid ultrasound would be indicated in the presence of a thyroid nodule.

Discuss (8) Improve

Next question

Pregnancy: thyroid problems

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link Report broken link

Score: **60.2%**

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Question 109 of 217 ✗

A 68-year-old man is admitted to the medical assessment unit with a 2-week history of lethargy, nausea, and mild confusion. He has a past medical history of small cell lung cancer diagnosed 3 months ago and is currently receiving chemotherapy. On examination, he is alert, oriented to time and place, and has no focal neurological deficits. His vital signs are stable with a blood pressure of 132/78 mmHg. There is no evidence of peripheral oedema or signs of dehydration. His blood results are as follows:

Na+	118 mmol/L	(135-145)
K+	4.2 mmol/L	(3.5-5.0)
Urea	5.1 mmol/L	(2.5-7.5)
Creatinine	76 µmol/L	(60-110)
Glucose	5.4 mmol/L	(3.5-5.5)
Serum osmolality	245 mOsm/kg	(275-295)
Urine osmolality	520 mOsm/kg	(50-1200)
Urine Na+	68 mmol/L	(variable)

Which of the following is the most appropriate initial management for this patient?

🟥	3% hypertonic saline infusion	10%
🟩	Fluid restriction to 800 mL/day	69%
	Normal saline (0.9%) infusion	8%
	Tolvaptan administration	5%
	Demeclocycline therapy	8%

Fluid restriction should be used in euvolemic and hypervolemic hyponatraemic patients who don't have severe symptoms

Important for me Less important

Fluid restriction to 800 mL/day is the most appropriate initial management for this patient. The clinical scenario describes a patient with chronic, moderate to severe hyponatraemia (Na+ 118 mmol/L) without severe neurological symptoms. The patient has mild confusion but is alert and oriented with no focal neurological deficits, suggesting the hyponatraemia is chronic rather than acute. The laboratory findings are consistent with syndrome of inappropriate antidiuretic hormone secretion (SIADH), which is a common paraneoplastic manifestation of small cell lung cancer. The diagnosis of SIADH is supported by hyponatraemia with low serum osmolality, inappropriately concentrated urine (high urine osmolality), and high urinary sodium excretion in a euvolemic patient. The patient's clinical examination shows no signs of volume depletion or overload, and normal renal function, further supporting SIADH. In euvolemic hyponatraemia due to SIADH without severe symptoms, fluid restriction is the first-line treatment to reduce free water intake and allow gradual correction of serum sodium levels.

3% hypertonic saline infusion is not appropriate in this case as it is reserved for patients with severe symptomatic hyponatraemia presenting with seizures, decreased consciousness (GCS <8), or other severe neurological symptoms. This patient has only mild confusion and is alert and oriented, so hypertonic saline would risk overly rapid correction and potential osmotic demyelination syndrome.

Normal saline (0.9%) infusion would be inappropriate for this patient with SIADH as it may worsen hyponatraemia. In SIADH, the kidneys continue to retain free water while excreting sodium, so isotonic saline can paradoxically lower serum sodium further. Normal saline is more appropriate for hypovolemic hyponatraemia, which this patient does not have.

Tolvaptan administration, a vasopressin receptor antagonist, is generally considered a second-line treatment for SIADH when fluid restriction is ineffective or impractical. It should not be used as first-line therapy due to potential side effects including hepatotoxicity and the risk of overly rapid correction of sodium levels. It is also expensive and requires careful monitoring.

Demeclocycline therapy is another second-line agent for chronic SIADH that is refractory to fluid restriction. It works by inducing nephrogenic diabetes insipidus, thereby reducing the kidney's response to ADH. However, it has a slow onset of action (3-4 days) and potential side effects including nephrotoxicity and photosensitivity. It would not be the most appropriate initial management in this case.

👍 👎 Discuss Improve

Next question

Hyponatraemia: treatment ★

Untreated, severe hyponatraemia may result in cerebral oedema, which in turn can cause brain herniation. It is therefore important to promptly identify and treat hyponatremia appropriately. However, the particular management for each patient is based on a number of factors listed below. Further to this, over-rapid correction may result in osmotic demyelination syndrome.

Principles

Management of hyponatremia is complicated and primarily based on the following parameters:

- duration of hyponatremia: is it acute or chronic?
    - acute: develops over a period of < 48 hours
    - chronic: develops over a period > 48 hours
- the severity of hyponatremia: what is the sodium level?
    - mild: 130-134 mmol/L
    - moderate: 120-129 mmol/L
    - severe: < 120 mmol/L
- symptoms: is the patient symptomatic?
    - patients with mild hyponatraemia may be symptomatic
    - early symptoms may include: headache, lethargy, nausea, vomiting, dizziness, confusion, and muscle cramps

    - late symptoms may include: seizures, coma, and respiratory arrest
- the suspected aetiology of the hyponatraemia:
    - hypovolemic hyponatraemia/clinically dehydrated: diuretic stage of renal failure, diuretics, Addisonian crisis
    - euvolemic hyponatraemia: SIADH
    - hypervolaemic hyponatraemia: heart failure, liver failure, nephrotic syndrome

Management

Initial steps in all patients

- exclude a spurious result (e.g. blood taken from a drip arm)
- review medications that may cause hyponatraemia

Chronic hyponatreamia without severe symptoms

If a hypovolemic cause is suspected

- normal, i.e. isotonic, saline (0.9% NaCl)
- this may sometimes be given as a trial
    - if the serum sodium rises this supports a diagnosis of hypovolemic hyponatraemia
    - if the serum sodium falls an alternative diagnosis such as SIADH is likely

If a euvolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider medications:
    - demeclocycline
    - vaptans (see below)

If a hypervolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider loop diuretics
- consider vaptans

Acute hyponatreamia with severe symptoms

Patients with acute, severe (<120 mmol/L) or symptomatic hyponatraemia require close monitoring, preferably in an HDU or above setting.

Hypertonic saline (typically 3% NaCl) is used to correct the sodium level more quickly than would be done in patients with chronic hyponatraemia.

Notes on specific medications

Vasopressin/ADH receptor antagonists (vaptans):

- these act primarily on V2 receptors - antagonism of V2 receptors results in selective water diuresis, sparing the electrolytes
- They should be avoided in patients who have hypovolemic hyponatremia
- Vasopression/ADH receptor antagonists can stimulate the **thirst** receptors leading to the desire to drink free water. They can be **hepatotoxic** in patients with underlying liver disease.

Complications of treatment

Osmotic demyelination syndrome (central pontine myelinolysis)

- can occur due to over-correction of severe hyponatremia
- pathophysiology:
    - thought to develop secondary to astrocyte (and possibly oligodendrocyte) apoptosis
    - astrocytes and oligodendrocytes (cells of the glial syncytium) are crucial for normal myelination
    - chronic hyponatraemia → loss of osmotically active organic osmolytes (such as myoinositol, glutamate, glutamine) from astrocytes. These provide protection against cerebral oedema
    - organic osmolytes cannot be replaced quickly enough when the brain volume begins to shrink in response to the correction of hyponatraemia

    - the dehydrated astrocytes and oligodendrocytes undergo apoptosis or other forms of injury → demyelination
- to avoid this, Na+ levels are only raised by 4 to 6 mmol/l in a 24-hour period
- symptoms usually occur after 2 days and are usually irreversible: dysarthria, dysphagia, paraparesis or quadriparesis, seizures, confusion, and coma
- patients are awake but are unable to move or verbally communicate, also called '**Locked-in syndrome**'

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 24 👎 28

2017 Hyponatraemia â€“ presentations and management

Suggest link Report broken link

Score: **59.6%**

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A 19-year-old woman presents to her GP with a 7 month history of weight loss, diarrhoea and palpitations. The diarrhoea is normal colour and over the last three months she has had roughly 2-3 bowel motions per day. The heart palpitations occur randomly throughout the day and night. She has also noticed that she has recently been getting episodes of feeling very hot and sweaty. She has no other past medical history and her only family history is a mother who has Hashimotos thyroiditis.

On examination, the patient is sweaty and her blood pressure is 130/80 mmHg, pulse is 102 bpm and regular, respiratory rate is 16/min and her oxygen SATs are 98% on air.

Blood tests are performed and reveal:

Hb	135 g/l
Platelets	220 * 10^9/l
WBC	7.1 * 10^9/l
Na^+	139 mmol/l
K^+	3.9 mmol/l
Urea	5.1 mmol/l
Creatinine	60 µmol/l
Free thyroxine (T4)	28 pmol/l
Thyroid stimulating hormone (TSH)	0.08 mu/l

A thyroid radioisotope scan is performed and reveals a globally reduced uptake.

What is the most likely diagnosis?

○ Graves disease ×

○ Thyrotoxicosis factitia ×

○ Hashimotos disease ×

● De Quervains thyroiditis ×

○ Atrophic thyroiditis ×

Submit answer

Reference ranges ∨

Score: **59.6%**

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🟩	Thyrotoxicosis factitia	23%
	Hashimotos disease	19%
🟥	De Quervains thyroiditis	35%
	Atrophic thyroiditis	6%

The most like likely diagnosis in this case is thyrotoxicosis factitia. This is evidenced by the reduced thyroid uptake on radioisotope scanning, along with the fact she may have easy access to thyroxine he mother would be taking it for her Hashimotos thyroiditis. De Quervains thyroiditis can present with symptoms similar to factitious thyroiditis and a decreased uptake on radioisotope scan, however, the fact the symptom shave lasted for 7 months makes this diagnosis unlikely.

👍 👎 Discuss (9) Improve

Next question

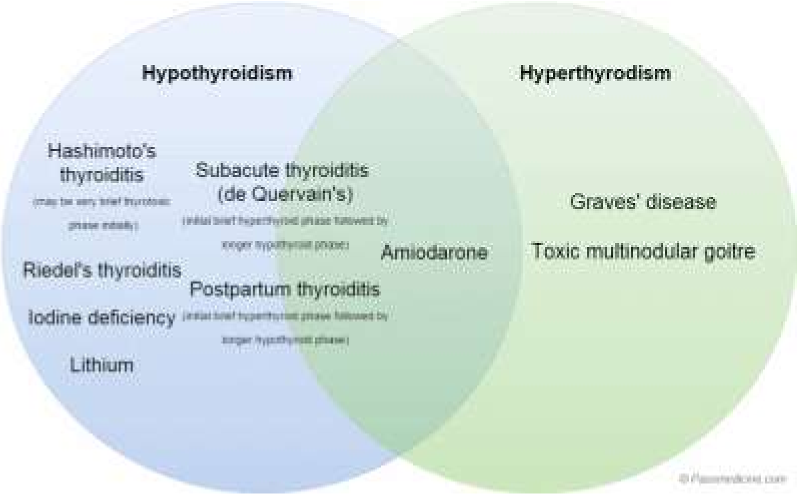
Thyrotoxicosis: causes and investigation ★

Graves' disease accounts for around 50-60% of cases of thyrotoxicosis.

Causes
- Graves' disease
- toxic nodular goitre
- acute phase of subacute (de Quervain's) thyroiditis
- acute phase of post-partum thyroiditis
- acute phase of Hashimoto's thyroiditis (later results in hypothyroidism)
- amiodarone therapy
- contrast
    - rare, more commonly occurs in elderly patients with pre-existing thyroid disease (e.g. multinodular goitre, Graves')
    - patients with existing thyrotoxicosis should not receive iodinated contrast medium

    - administration of iodinated contrast results in a large iodine load to the thyroid → hyperthyroidism developing over 2-12 weeks

Investigation
- TSH down, T4 and T3 up
- thyroid autoantibodies
- other investigations are not routinely done but includes isotope scanning


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
© Passmedicine.com

Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 2 👎 4

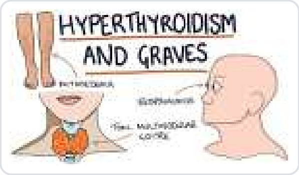
Hyperthyroidism guidelines

Royal College of Physicians 👍 7 👎 3

2017 Thyrotoxicosis â€“ investigation and management

Suggest link Report broken link

Media


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 2 👎 0

ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 1 👎 0

Clinical features

Thyroid disease

Podmedics - YouTube 👍 0 👎 0

Report broken media

Score: **59.1%**

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You are seeing a 50-year-old lady with type 2 diabetes mellitus in the outpatient clinic. She has a past medical history of gastritis, moderate left ventricular dysfunction and chronic obstructive pulmonary disease. She is currently on metformin and gliclazide. Since last review she has gained 5kg in weight and her HbA1c has deteriorated to 70 mmol/mol from 62 mmol/mol. Body mass index today in clinic is 33 kg/m².

Recent blood tests are as follows:

Na^+	141 mmol/l
K^+	3.9 mmol/l
Urea	6 mmol/l
Creatinine	140 µmol/l

She was unable to previously tolerate liraglutide due to nausea and vomiting. What would be the best alteration to her therapy?

- ● Empagliflozin (SGLT-2 inhibitor) ✕
- ○ Add insulin ✕
- ○ Add pioglitazone ✕
- ○ Increase dose of metformin ✕
- ○ Increase dose of gliclazide ✕

Submit answer

Reference ranges

Score: **59.1%**

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She was unable to previously tolerate liraglutide due to nausea and vomiting. What would be the best alteration to her therapy?

Empagliflozin (SGLT-2 inhibitor)	92%
Add insulin	5%
Add pioglitazone	2%
Increase dose of metformin	1%
Increase dose of gliclazide	0%

SGLT inhibitors have the advantage of improving glycaemic control/HbA1c and having beneficial effects on weight. This is because their mode of action is independent of insulin release. They act upon the SGLT-2 receptors in the kidney and lead to increased loss of glucose in the urine.

👍 | 👎 | Discuss (4) | Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

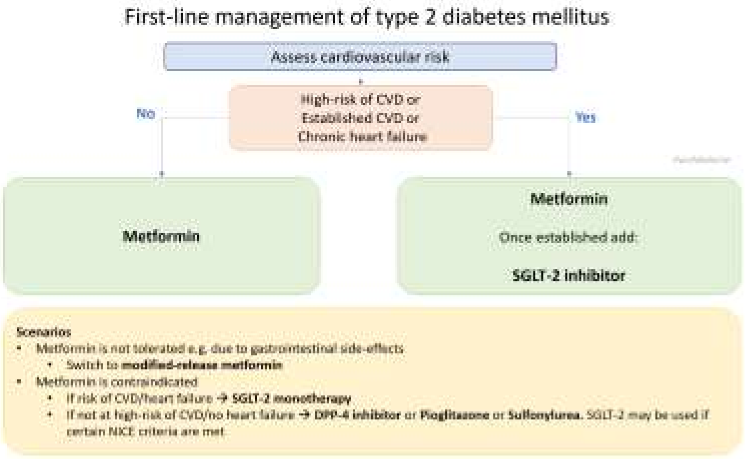
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

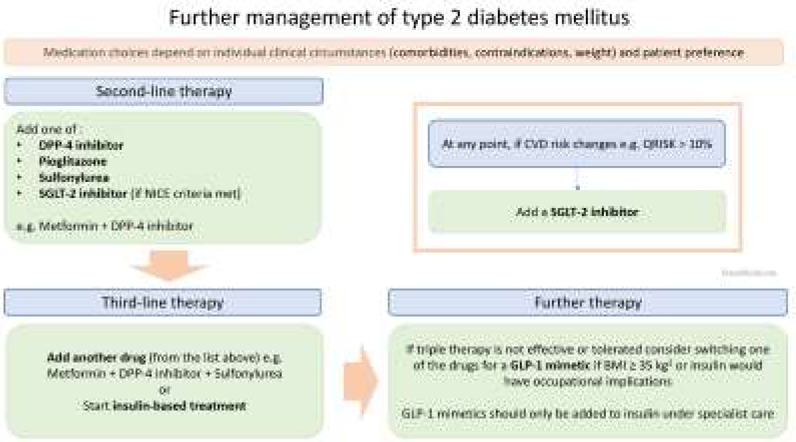
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:

  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus

Medication choices depend on individual clinical circumstances **(comorbidities, contraindications, weight) and patient preference**

Second-line therapy

Add one of :
- **DPP-4 inhibitor**
- **Pioglitazone**
- **Sulfonylurea**
- **SGLT-2 inhibitor** (if NICE criteria met)

e.g. Metformin + DPP-4 inhibitor

At any point, if CVD risk changes e.g. QRISK > 10%

Add a **SGLT-2 inhibitor**

Third-line therapy

Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start **insulin-based treatment**

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a **GLP-1 mimetic** if BMI ≥ 35 kg^2 or insulin would have occupational implications

GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

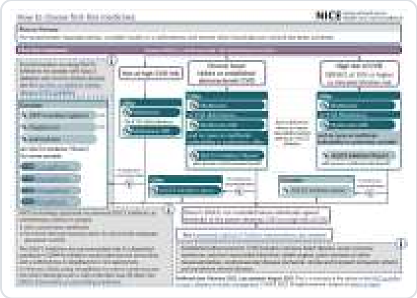
Starting insulin

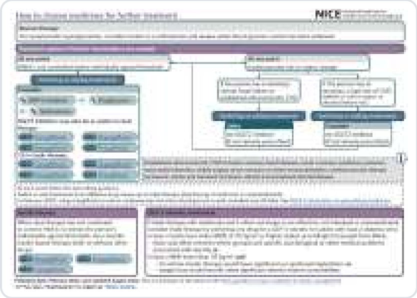
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

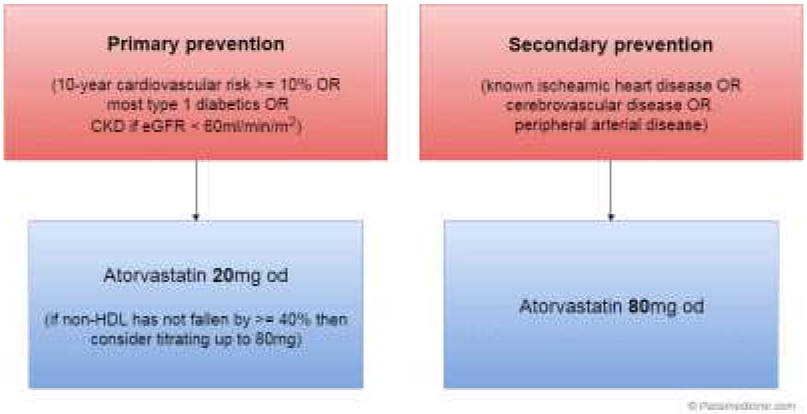
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

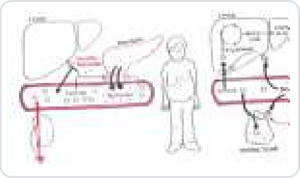
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

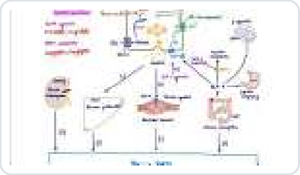
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **59.5%**

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A 60-year- old female presented with a six month history of polyuria, polydipsia and generalised aches and pains.

She is a known hypertensive for ten years and is taking bendroflumethiazide 2.5 mg daily. She has been taking calcium and vitamin D supplements for the last two years as she has a strong family history of osteoporosis.

On examination, her pulse rate is 80 beats per minute and her blood pressure is 150/90 mmHg. Cardiovascular, respiratory and abdominal examination were normal.

Investigations reveal:

Serum sodium	130 mmol/L
Serum potassium	3.1 mmol/L
Serum urea	7.7 mmol/L
Serum creatinine	88 mol/L
Serum corrected calcium	2.9 mmol/L
Phosphate	0.8 mmol/L
PTH	4.5 pmol/L (0.9-5.4)
Urinalysis	glycosuria ++

What is the most likely cause of this ladys symptoms?

○ Primary hyperparathyroidism ✕

○ Vitamin D excess ✕

○ Bendroflumethizide induced hypercalcaemia ✕

○ Familial hypocalciuric hypercalcaemia ×

○ Diabetes mellitus ×

Submit answer

Reference ranges ﹀

Score: **59.5%**

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Dr. Jamil Alrajhi

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← Question 112 of 217 ✗ →

A 60-year- old female presented with a six month history of polyuria, polydipsia and generalised aches and pains.

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Serum corrected calcium	2.9 mmol/L
Phosphate	0.8 mmol/L
PTH	4.5 pmol/L (0.9-5.4)
Urinalysis	glycosuria ++

What is the most likely cause of this ladys symptoms?

🟩	Primary hyperparathyroidism	28%
	Vitamin D excess	7%
🟥	Bendroflumethizide induced hypercalcaemia	39%

Familial hypocalciuric hypercalcaemia 18%

Diabetes mellitus 7%

The PTH level in primary hyperparathyroidism may be normal

Important for me Less important

This lady has hypercalcaemia which may be due to bendroflumethiazide or vitamin D excess, but the PTH level is inappropriately normal in the context of hypercalcaemia which indicates primary hyperparathyroidism rather than any other cause of hypercalcaemia in which the PTH would be suppressed by homeostatic mechanisms.

In addition, the phosphate level is low which is typical of primary hyperparathyroidism. The glycosuria is a distractor.

Discuss (26) Improve

Next question

Primary hyperparathyroidism ★

Primary hyperparathyroidism is caused by excess secretion of PTH resulting in hypercalcaemia. It is the most common cause of hypercalcaemia in outpatients and is often diagnosed following an incidental finding of an elevated serum calcium concentration. In 85% of cases a parathyroid adenoma is responsible.

Causes of primary hyperparathyroidism

- 85%: solitary adenoma
- 10%: hyperplasia
- 4%: multiple adenoma
- 1%: carcinoma

Around 80% of patients are asymptomatic and are diagnosed on routine blood tests. The symptomatic features of primary hyperparathyroidism may be remembered by the mnemonic: 'bones, stones, abdominal groans and psychic moans':

- polydipsia, polyuria
- depression
- anorexia, nausea, constipation
- peptic ulceration
- pancreatitis
- bone pain/fracture
- renal stones
- hypertension

Associations

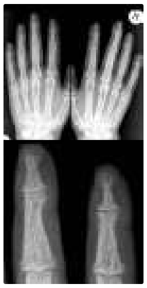
- hypertension
- multiple endocrine neoplasia: MEN I and II

Investigations

- bloods
  - raised calcium, low phosphate
  - PTH may be raised or (inappropriately, given the raised calcium) normal
- technetium-MIBI subtraction scan
- x-ray findings
  - pepperpot skull
  - osteitis fibrosa cystica

Treatment

- the definitive management is total parathyroidectomy
- conservative management may be offered if the calcium level is less than 0.25 mmol/L above the upper limit of normal AND the patient is > 50 years AND there is no evidence of end-organ damage
- patients not suitable for surgery may be treated with cinacalcet, a calcimimetic
  - a calcimimetic 'mimics' the action of calcium on tissues by allosteric activation of the calcium-sensing receptor



+ Q 123

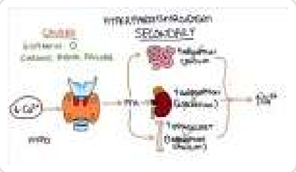
Next question

Textbooks

High-yield textbook

Extended textbook

Media


HYPERPARATHYROIDISM
SECONDARY

Understanding Hyperparathyroidism

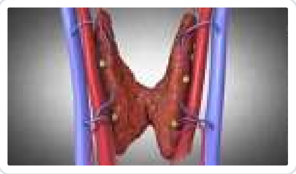
Zero to Finals - YouTube

13 0

ENDOCRINOLOGY
HYPERPARATHYROIDISM

Hyperparathyroidism and the different types, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 2 👎 0



Parathyroid Glands and Hyperparathyroidism

Norman Parathyroid Center - YouTube 👍 2 👎 1

Report broken media

Score: **58.9%**

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You are seeing a 58-year-old man in clinic who has been referred by his GP with resistant hypertension despite treatment with ramipril, amlodipine, indapamide and bisoprolol. He looks comfortable at rest.

Observations are as follows: temperature 36.5ºC, blood pressure 182/125 mmHg, heart rate 88/min, respiratory rate 16/min, saturations 97% on air

Investigations are as follows:

Na^+	148 mmol/l
K^+	2.9 mmol/l
Urea	6.5 mmol/l
Creatinine	92 µmol/l

Renin	Low
Aldosterone (supine)	High
Aldosterone (prolonged standing)	Increase from supine levels

Blood gases:

PaO2	11.2kPa
PaCO2	5.2kPa
pH	7.49
HC03-	32 mmol/l
BE	+4

What is the single most likely diagnosis?

Adrenal hyperplasia 45%

Conn's adenoma	46%
Malignant hypertension	1%
Phaeochromocytoma	2%
Renal artery stenosis	6%

All of the above diagnoses could cause hypertension. This patient has a hypokalaemic alkalosis and low renin. The differentials are therefore Conn's adenoma and adrenal hyperplasia. To differentiate between the two look at the aldosterone on standing. In this case, the aldosterone increases on prolonged standing, therefore, the diagnosis is adrenal hyperplasia. In Conn's adenoma, the aldosterone would stay the same or drop on standing.

In malignant hypertension and renal artery stenosis, the renin would be high.

👍 👎 💬 Discuss (9) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes

- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism

- adrenal carcinoma

Features

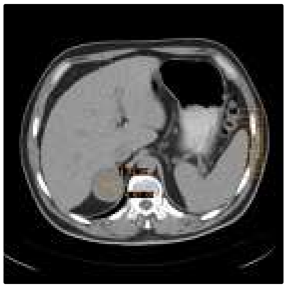
- **hypertension**
    - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
    - e.g. muscle weakness
    - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
    - hypertension with hypokalemia
    - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
    - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
    - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

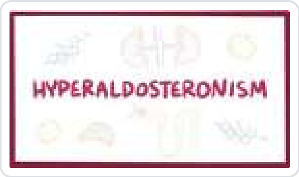
The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

Suggest link

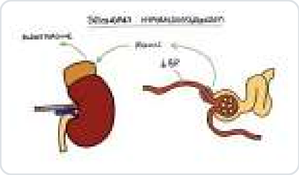
Report broken link

Media



Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

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Score: **58.4%**

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113	✗

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A 68-year-old gentleman presents with severe abdominal pain. He has a past medical history of depression and type 2 diabetes. His medications include; levemir, sitagliptin and gliclazide. He denies smoking, alcohol or illicit drug use. He also claims he has been taking all his diabetic medications.

On examination he is overweight. He has dry mucus membranes. He has generalised abdominal pain with no rebound or guarding. His bowel sounds are present and normal. His blood pressure is 101/76 mmHg, his pulse rate is 113 beats per minute and his temperature is 37.8°C.

Investigations;

Erect Chest X Ray	Clear
Ultrasound scan of the abdomen	Biliary duct 3mm Normal appearance of gallbladder Diffuse increase echogenicity of liver, normal size Pancreas no visible abnormality

Hb	134 g/dL
WCC	13.1 *10^9/l
Platelets	234 *10^9/l
MCV	89 fL

Sodium	148 mmol/L
Potassium	5.6 mmol/L
Creatinine	165 µmol/L
Urea	10.4 mmol/L
Alkaline Phosphatase	76 IU/L
Alanine Transaminase	42 IU/L
Gamma-glutamyl transpeptidase	60 IU/L

Amylase	1378 IU/L
Glucose	38 mmol/l
Urinalysis	WCC + RBC ve Ketones +

What is the likely underlying diagnosis?

- ○ Diabetic ketoacidosis ✕
- ○ Perforated small bowel ✕
- ○ Drug induced pancreatitis ✕
- ○ Acute hepatitis ✕
- ○ Alcohol induced pancreatitis ✕

Submit answer

Reference ranges ⌄

Score: **58.4%**

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Question 114 of 217 ✗

A 68-year-old gentleman presents with severe abdominal pain. He has a past medical history of depression and type 2 diabetes. His medications include; levemir, sitagliptin and gliclazide. He denies smoking, alcohol or illicit drug use. He also claims he has been taking all his diabetic medications.

On examination he is overweight. He has dry mucus membranes. He has generalised abdominal pain with no rebound or guarding. His bowel sounds are present and normal. His blood pressure is 101/76 mmHg, his pulse rate is 113 beats per minute and his temperature is 37.8°C.

Investigations;

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Amylase	1378 IU/L
Glucose	38 mmol/l
Urinalysis	WCC + RBC ve Ketones +

What is the likely underlying diagnosis?

	Diabetic ketoacidosis	24%
	Perforated small bowel	1%
🟩	Drug induced pancreatitis	68%
	Acute hepatitis	1%
🟥	Alcohol induced pancreatitis	5%

Answer: Sitagliptiin induced pancreatitis.

Patients with perforated small bowel or DKA can present with a raised amylase. The level suggests the cause is most likely pancreatitis. A perforated small bowel would more likely show free gas on a Chest X ray, diminished bowel sounds and guarding. Diabetic ketoacidosis would show more ketones on urinalysis and is rare, although still possible, in type 2 diabetics.

The gentleman has no evidence of gallstones or biliary obstruction. This combined with a normal liver function tests makes gallstone unlikely. The patient denies alcohol intake and non-alcoholic fatty liver can produce the appearances on ultrasound scan.

Both dipeptidyl peptidase 4 inhibitors (sitagliptin) and glucagon like peptide-1 agonists have been linked to pancreatitis. They are still both under investigation but is to be discontinued in the event of an episode of pancreatitis.

Discuss (10) | Improve

Next question

Diabetes mellitus: GLP-1 drugs

A number of drugs to treat diabetes mellitus have become available in recent years. Much research has focused around the role of glucagon-like peptide-1 (GLP-1), a hormone released by the small intestine in response to an oral glucose load

Whilst it is well known that insulin resistance and insufficient B-cell compensation occur other effects are also seen in type 2 diabetes mellitus (T2DM). In normal physiology an oral glucose load results in a greater release of insulin than if the same load is given intravenously - this known as the incretin effect. This effect is largely mediated by GLP-1 and is known to be decreased in T2DM.

Increasing GLP-1 levels, either by the administration of an analogue (glucagon-like peptide-1, GLP-1 mimetics, e.g. exenatide) or inhibiting its breakdown (dipeptidyl peptidase-4 ,DPP-4 inhibitors - the gliptins), is therefore the target of two recent classes of drug.

Glucagon-like peptide-1 (GLP-1) mimetics (e.g. exenatide)

Exenatide is an example of a glucagon-like peptide-1 (GLP-1) mimetic. These drugs increase insulin secretion and inhibit glucagon secretion. One of the major advances of GLP-1 mimetics is that they typically result in weight loss, in contrast to many medications such as insulin, sulfonylureas and thiazolidinediones. They are sometimes used in combination with insulin in T2DM to minimise weight gain.

Exenatide must be given by subcutaneous injection within 60 minutes before the morning and evening meals. It should not be given after a meal.

Liraglutide is the other GLP-1 mimetic currently available. One the main advantages of liraglutide over exenatide is that it only needs to be given once a day.

Both exenatide and liraglutide may be combined with metformin and a sulfonylurea. Standard release exenatide is also licensed to be used with basal insulin alone or with metformin. Please see the BNF for a more complete list of licensed indications.

NICE state the following:

> *Consider adding exenatide to metformin and a sulfonylurea if:*
> - *BMI >= 35 kg/m² in people of European descent and there are problems associated with high weight, or*
> - *BMI < 35 kg/m² and insulin is unacceptable because of occupational implications or weight loss would benefit other comorbidities.*

NICE like patients to have achieved a > 11 mmol/mol (1%) reduction in HbA1c and 3% weight loss after 6 months to justify the ongoing prescription of GLP-1 mimetics.

The major adverse effect of GLP-1 mimetics is nausea and vomiting. The Medicines and Healthcare products Regulatory Agency has issued specific warnings on the use of exenatide, reporting that is has been linked to severe pancreatitis in some patients.

Dipeptidyl peptidase-4 (DPP-4) inhibitors (e.g. Vildagliptin, sitagliptin)

Key points

- dipeptidyl peptidase-4, DPP-4 inhibitors increase levels of incretins (GLP-1 and GIP) by decreasing their peripheral breakdown

- oral preparation
- trials to date show that the drugs are relatively well tolerated with no increased incidence of hypoglycaemia
- do not cause weight gain

NICE guidelines on DPP-4 inhibitors

- NICE suggest that a DPP-4 inhibitor might be preferable to a thiazolidinedione if further weight gain would cause significant problems, a thiazolidinedione is contraindicated or the person has had a poor response to a thiazolidinedione

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

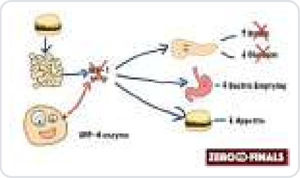
Links

NICE 👍 20 👎 14

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Media



How does sitagliptin work? DPP-4 inhibitors and GLP-1 mimetics

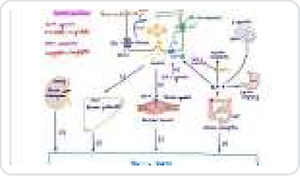
Zero to Finals - YouTube 👍 2 👎 0

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 1 👎 0

Glucagon-like Peptide (GLP-1) and the treatment of diabetes

Medicosis Perfectionalis - YouTube 👍 2 👎 1



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 0 👎 1

Report broken media

Score: **57.9%**

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A 47-year-old man attended his GP after checking his blood pressure at the local pharmacy. When he had it checked it was 179/102 mmHg. The GP confirmed it was high in the surgery at 186/103 mmHg. He started him on ramipril 2.5mg and titrated up to the dose to 10mg over the next few weeks. His repeat measurements showed consistently high readings so the GP added amlodipine, which had very little effect despite being tolerated at the maximum dose. After failing to get an adequate response with the addition of a third agent the GP referred the patient to the endocrine clinic.

Observations showed a blood pressure of 190/105 mmHg and a heart rate of 98 beats per minute. On examination, the man was thin with a body mass index of 23 kg/m². His apex was diffuse and displaced with normal heart sounds. The chest was clear and abdomen was soft and non-tender with no evidence of masses or renal bruits. He was noted to have a hard, painless nodule over the thyroid gland.

The 24 hour urinary catecholamines were raised and further investigations confirmed phaeochromocytoma. He was treated medically with an alpha blocker then beta blocker whilst awaiting surgery. In this period he had further investigation into the thyroid nodule, which was a cold nodule on radionucleotide scanning.

Which type of thyroid cancer would you expect this to be histologically?

Papillary	7%
Follicular	5%
Anaplastic	2%
Lymphoma	1%
Medullary	86%

This question tests your knowledge of multiple endocrine neoplasias (MEN). This patient could have MEN Type IIa or IIb, which both include phaeochromocytoma and medullary thyroid cancer.

Of all thyroid cancers, medullary thyroid cancer accounts for approximately 5% (and of these, 80% will be sporadic rather than associated with MEN).

👍 👎 Discuss (4) Improve

Next question

Thyroid cancer ★

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy

- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature

Type	Notes
	• Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

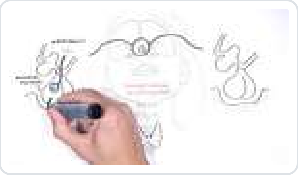
Suggest link Report broken link

Media

THYROID
CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0



Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **58.3%**

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Question 116 of 217 ✓

A 28-year-old male with a history of epilepsy, for which he is taking carbamazepine and has not had any seizures for the last two years, presents with irritability and nausea for the last 2 weeks. His girlfriend says that he is often confused and seems to be lost most of the time. He takes alcohol occasionally and smokes ten to twelve cigarettes per day.

On examination, he is irritable but conscious and alert. Clinical examination revealed eczema over the face, shins and extensor surfaces of the forearms and a tattoo on the right shoulder. There was no evidence of any peripheral oedema.

Lab reports were as follows:

Hb	150 g/l
MCV	81 fl
MCH	31 pg
WBC	9 * 10^9/l
Plt	250 * 10^9/l
Urea	3.2 mmol/l
Creatinine	75 µmol/l
9:00 am Cortisol	345 nmol/l (170 700 nmol/l)
TSH	2.4 mU/l
Total T4	102 nmol/l (68 174 nmol/l)
Na+	119 mmol/l
K+	4.2 mmol/l

Which of the following would be the most appropriate initial management option?

Fluid restriction to 500 - 1000 ml daily 58%

Demeclocycline 600 - 1200 mg daily	7%
IV hypertonic saline	32%
Intranasal desmopressin twice daily	2%
Hydrochlorothiazide 12.5 mg daily	1%

The diagnosis in the scenario described above is SIADH secondary to carbamazepine treatment. The findings of eczema are coincidental and have no bearing on the diagnosis. However, in the management the presence of skin lesions such as eczema may pose difficulty in opting for the use of demeclocycline which may cause photosensitive rashes.

The initial treatment option for SIADH is by fluid restriction which, under most circumstances, will correct the biochemical abnormalities in almost every case.

Demeclocycline is indicated if fluid restriction is poorly tolerated or ineffective.

Hypertonic saline is indicated only when the syndrome is very severe, acute and symptomatic.

Intranasal desmopressin is used for the management of diabetes insipidus, and thiazide diuretics are only rarely used as alternative agents in diabetes insipidus.

The causes of SIADH are as follows

1. Tumours
    - Small cell CA of the lung
    - Prostate
    - Thymus
    - Pancreas
    - Lymphoma

2. Pulmonary Lesions
    - Pneumonia

- Tuberculosis
- Lung abscess

3. CNS Causes
- Meningitis
- Tumours
- Head injury
- Subdural haematoma
- Cerebral abscess
- SLE
- Vasculitis

4. Metabolic Causes
- Alcohol withdrawal
- Porphyrias

5. Drugs
- Carbamazepine
- Chlorpropamide
- Cyclophosphamide
- Vincristine
- Phenothiazines

👍 | 👎 | Discuss (12) | Improve

Next question

Hyponatraemia: treatment ★

Untreated, severe hyponatraemia may result in cerebral oedema, which in turn can cause brain herniation. It is therefore important to promptly identify and treat hyponatremia appropriately. However, the particular management for each patient is based on a number of factors listed below. Further to this, over-rapid correction may result in osmotic demyelination syndrome.

Principles

Management of hyponatremia is complicated and primarily based on the following parameters:

- duration of hyponatremia: is it acute or chronic?
  - acute: develops over a period of < 48 hours
  - chronic: develops over a period > 48 hours
- the severity of hyponatremia: what is the sodium level?
  - mild: 130-134 mmol/L
  - moderate: 120-129 mmol/L
  - severe: < 120 mmol/L
- symptoms: is the patient symptomatic?
  - patients with mild hyponatraemia may be symptomatic
  - early symptoms may include: headache, lethargy, nausea, vomiting, dizziness, confusion, and muscle cramps
  - late symptoms may include: seizures, coma, and respiratory arrest
- the suspected aetiology of the hyponatraemia:
  - hypovolemic hyponatraemia/clinically dehydrated: diuretic stage of renal failure, diuretics, Addisonian crisis
  - euvolemic hyponatraemia: SIADH
  - hypervolaemic hyponatraemia: heart failure, liver failure, nephrotic syndrome

Management

Initial steps in all patients

- exclude a spurious result (e.g. blood taken from a drip arm)
- review medications that may cause hyponatraemia

Chronic hyponatreamia without severe symptoms

If a hypovolemic cause is suspected

- normal, i.e. isotonic, saline (0.9% NaCl)
- this may sometimes be given as a trial

    - if the serum sodium rises this supports a diagnosis of hypovolemic hyponatraemia
    - if the serum sodium falls an alternative diagnosis such as SIADH is likely

If a euvolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider medications:
    - demeclocycline
    - vaptans (see below)

If a hypervolemic cause is suspected

- fluid restrict to 500-1000 mL/day
- consider loop diuretics
- consider vaptans

Acute hyponatreamia with severe symptoms

Patients with acute, severe (<120 mmol/L) or symptomatic hyponatraemia require close monitoring, preferably in an HDU or above setting.

Hypertonic saline (typically 3% NaCl) is used to correct the sodium level more quickly than would be done in patients with chronic hyponatraemia.

Notes on specific medications

Vasopressin/ADH receptor antagonists (vaptans):

- these act primarily on V2 receptors - antagonism of V2 receptors results in selective water diuresis, sparing the electrolytes
- They should be avoided in patients who have hypovolemic hyponatremia
- Vasopression/ADH receptor antagonists can stimulate the **thirst** receptors leading to the desire to drink free water. They can be **hepatotoxic** in patients with underlying liver disease.

Complications of treatment

Osmotic demyelination syndrome (central pontine myelinolysis)

- can occur due to over-correction of severe hyponatremia
- pathophysiology:
    - thought to develop secondary to astrocyte (and possibly oligodendrocyte) apoptosis
    - astrocytes and oligodendrocytes (cells of the glial syncytium) are crucial for normal myelination
    - chronic hyponatraemia → loss of osmotically active organic osmolytes (such as myoinositol, glutamate, glutamine) from astrocytes. These provide protection against cerebral oedema
    - organic osmolytes cannot be replaced quickly enough when the brain volume begins to shrink in response to the correction of hyponatraemia
    - the dehydrated astrocytes and oligodendrocytes undergo apoptosis or other forms of injury → demyelination
- to avoid this, Na+ levels are only raised by 4 to 6 mmol/l in a 24-hour period
- symptoms usually occur after 2 days and are usually irreversible: dysarthria, dysphagia, paraparesis or quadriparesis, seizures, confusion, and coma
- patients are awake but are unable to move or verbally communicate, also called '**Locked-in syndrome**'

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 24 👎 28

2017 Hyponatraemia â€“ presentations and management

Suggest link Report broken link

Score: **58.6%**

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57	✓
58	✗
59	✗
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107	✗
108	✓
109	✗
110	✗
111	✓
112	✗
113	✗
114	✗
115	✓
116	✓

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A 52-year-old female presents with galactorrhoea. On systematic enquiry she reveals that she has been feeling very fatigued recently. She has no past medical history and takes no regular medicines.

Blood results are as follows:

Hb	125 g/l	Na$^+$	132 mmol/l
Platelets	422 * 10^9/l	K$^+$	3.8 mmol/l
WBC	9.2 * 10^9/l	Urea	6.4 mmol/l
Neuts	6.2 * 10^9/l	Creatinine	41 µmol/l
Lymphs	2.4 * 10^9/l	CRP	4 mg/l
Prolactin	440 ng/dL (normal 5 - 40)		

What investigation will you perform?

- ○ MRI pituitary ×
- ○ Synacthen test ×
- ○ Thyroid function tests ×
- ○ Toxicology screen ×
- ○ No further investigations required ×

Submit answer

Reference ranges

Score: **0%**

1	-

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← Question 1 of 101 ✓ →

A 52-year-old female presents with galactorrhoea. On systematic enquiry she reveals that she has been feeling very fatigued recently. She has no past medical history and takes no regular medicines.

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Neuts	6.2 * 10^9/l	Creatinine	41 µmol/l
Lymphs	2.4 * 10^9/l	CRP	4 mg/l
Prolactin	440 ng/dL (normal 5 - 40)		

What investigation will you perform?

MRI pituitary	58%
Synacthen test	5%
Thyroid function tests	34%
Toxicology screen	1%
No further investigations required	3%

Primary hypothyroidism can cause hyperprolactinaemia. High leveles of thyrotrophin releasing hormone (TRH) stimulate prolactin release

Important for me Less important

The patient has galactorrhoea secondary to hyperprolactinaemia. From the history there is not much information to narrow down our differential diagnosis which is currently wide. Hypothyroidism is a very common condition and can cause hyperprolactinaemia. High levels of thyrotrophin releasing hormone (TRH) seen in primary hypothyroidism stimulate prolactin release. Hypothyroidism can also cause hyponatraemia as seen in this case. Thyroid function tests are therefore the best answer. A pregnancy test should also be considered.

The Synacthen test is used to assess for hypoadrenalism. Hypoadrenalism is not linked to hyperprolactinaemia.

Certain drugs can cause hyperprolactinaemia (e.g. dopamine antagonists). However a toxicology screen would not detect these agents.

Once the common causes of hyperprolactinaemia have been excluded (e.g. hypothyroidism, pregnancy, and drugs) then you should consider investigating for a pituitary adenoma with an MRI scan.

👍 👎 Discuss (5) Improve

Next question

Prolactin and galactorrhoea

Prolactin is secreted by the anterior pituitary gland with release being controlled by a wide variety of physiological factors. Dopamine acts as the primary prolactin releasing inhibitory factor and hence dopamine agonists such as bromocriptine may be used to control galactorrhoea. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia

Features of excess prolactin

- men: impotence, loss of libido, galactorrhoea
- women: amenorrhoea, galactorrhoea

Causes of raised prolactin

- prolactinoma
- pregnancy
- oestrogens
- physiological: stress, exercise, sleep
- acromegaly: 1/3 of patients
- polycystic ovarian syndrome
- primary hypothyroidism (due to thyrotrophin releasing hormone (TRH) stimulating prolactin release)

Drug causes of raised prolactin

- metoclopramide, domperidone
- phenothiazines
- haloperidol
- very rare: SSRIs, opioids

AI + Q 123

Next question

B I A

Textbooks

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Links

Patient.info 👍 11 👎 5

Galactorrhoea review

Suggest link Report broken link

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **100%**

1 ✓

A 77-year-old female is admitted to the acute medal unit with a chest infection. Her past medical history includes COPD and heart failure. Her current medications include salbutamol, tiotropium, bisoprolol and ramipril. On examination you note left basal crepitations. The JVP is at 2cm above the sternal angle. There is no peripheral oedema. Her blood pressure is 145/85 mmHg.

You note that her blood results are as follows:

Na^+	120 mmol/l
Urine osmolarity	400 mosmol/l
Urine sodium	50 mosmol/l
Urea	6.2 mmol/l
Creatinine	54 µmol/l
fT3	6.2 pmol/l (normal range 3.5 - 7.8)
fT4	6 pmol/l (normal range 9.0 - 25.0)
TSH	0.2 mU/l (normal range 0.4 - 4.0)
morning cortisol	normal

You water restrict the patient to 1.5 litres per day. On day 4 her bloods are reported as follows:

Na^+	118 mmol/l

How would you manage this patient?

○ Furosemide ×

○ Levothyroxine ×

○ Demeclocycline ×

○ Hypertonic saline (3%) ✕

○ Hypotonic saline (0.45%) ✕

Submit answer

Reference ranges ∨

Score: **100%**

1	✓
2	-

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Question 2 of 101 ✓

A 77-year-old female is admitted to the acute medal unit with a chest infection. Her past medical history includes COPD and heart failure. Her current medications include salbutamol, tiotropium, bisoprolol and ramipril. On examination you note left basal crepitations. The JVP is at 2cm above the sternal angle. There is no peripheral oedema. Her blood pressure is 145/85 mmHg.

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fT4	6 pmol/l (normal range 9.0 - 25.0)
TSH	0.2 mU/l (normal range 0.4 - 4.0)
morning cortisol	normal

You water restrict the patient to 1.5 litres per day. On day 4 her bloods are reported as follows:

Na^+	118 mmol/l

How would you manage this patient?

Furosemide	9%
Levothyroxine	11%
Demeclocycline	55%

Hypertonic saline (3%)	23%
Hypotonic saline (0.45%)	2%

If SIADH is resistant to fluid restriction then the drug demeclocycline can be used

Important for me Less important

The differential for hyponatraemia is wide. The fluid status of the patient is of paramount importance for identifying the cause. In this example, the patient is clearly euvolaemic. Causes of euvolaemic hyponatraemia include SIADH, hypothyroidism, and adrenal insufficiency. The serum and urine osmolarities, and urinary sodium are consistent with either of these diagnoses.

SIADH is effectively a diagnosis of exclusion - primary hypothyroidism and hypoadrenalism must be excluded to make the diagnosis.

In this case, the thyroid function tests are suggestive of euthyroid sick syndrome. This would not be an indication for initiating levothyroxine. Although hypothyroidism is a well-known cause of hyponatraemia euthyroid sick syndrome would not effect sodium balance. It is often stated that to make a diagnosis of SIADH relies upon 'normal thyroid function tests'. It is important to note that this is referring to primary hypothyroidism. Euthyroid sick syndrome does not preclude the diagnosis of SIADH.

The morning cortisol is normal which suggests that hypoadrenalism is not present.

Therefore the most likely diagnosis is SIADH secondary to a chest infection. The initial treatment for non-severe SIADH is fluid restriction.

This patient has been trialled with fluid restriction however the sodium has not improved and has in fact got worse. The next step in the management would be demeclocycline. Demeclocycline inhibits the effects of ADH on the renal tubules. It effectively causes a nephrogenic diabetes insipidus.

The patient's symptoms are not severe enough to warrant use of hypertonic saline. Hypertonic saline has risks associated with it, as rapid correction of serum Na can result in central pontine myelinolysis.

If the patient had been hypervolaemic then furosemide would be indicated.

Discuss (14) Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology
- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water balance
- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels
- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.
- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension, because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted,

leading to hyponatraemia.

Causes of SIADH

Category	Examples
Malignancy	• **small cell lung cancer** • also: pancreas, prostate
Neurological	• stroke • subarachnoid haemorrhage • subdural haemorrhage • meningitis/encephalitis/abscess
Infections	• tuberculosis • pneumonia
Drugs	• sulfonylureas* • SSRIs, tricyclics • carbamazepine • vincristine • cyclophosphamide
Other causes	• positive end-expiratory pressure (PEEP) • porphyrias

Investigations

- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.
- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management

- correction must be done slowly to avoid precipitating central pontine myelinolysis

- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

AI + Q 123

Next question

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Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **100%**

1	✓
2	✓

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Question 3 of 101

A 54-year-old woman attends with severe headaches. The headaches have been recurrent and are precipitated by exercise or drinking coffee. She has also felt intolerant to heat and is generally nauseated.

She has a past medical history of anxiety, constipation, and recurrent renal stones.

On examination, she is diaphoretic and tachycardic. There is no focal neurology. She has palpable cervical lymphadenopathy.

Heart rate 120 beats per minute, blood pressure 180/102, SpO2 98% room air, respiratory rate 18 breaths per minute, temperature 37.3°C.

Hb	120 g/L	Male: (135-180) Female: (115 - 160)
Platelets	222 * 10^9/L	(150 - 400)
WBC	6.5 * 10^9/L	(4.0 - 11.0)
Calcium	3.3 mmol/L	(2.1-2.6)
Thyroid-stimulating hormone (TSH)	4.5 mU/L	(0.5-5.5)
Free thyroxine (T4)	14 pmol/L	(9.0 - 18)
LH	10 IU/L	(5-25)
FSH	0.6 IU/L	(0.3-10)
Capillary blood glucose	5.4 mmol/L	(3.9-7.8)

Given the likely diagnosis, which of the following options should form part of the workup for this patient?

○ MRI head

○ MRI pancreas

○ Serum calcitonin

○ Skeletal survey ×

○ Ultrasound adrenals ×

Submit answer

Reference ranges

Score: **100%**

1 ✓
2 ✓
3 -

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FSH	0.6 IU/L	(0.3-10)
Capillary blood glucose	5.4 mmol/L	(3.9-7.8)

Given the likely diagnosis, which of the following options should form part of the workup for this patient?

🟥 MRI head	15%
MRI pancreas	7%
🟩 Serum calcitonin	51%

Skeletal survey	6%
Ultrasound adrenals	21%

MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer

Important for me Less important

Here we have a patient presenting with the classic triad of pheochromocytoma - hypertension, headaches, and sweating alongside cervical lymphadenopathy and hypercalcemia. The most common cause of hypercalcemia is primary hyperparathyroidism. The unifying diagnosis here is MEN type 2A. Up to 100% of patients with MEN type 2A have medullary thyroid cancer at some time in their life and so testing for this is essential.

Serum calcitonin is correct. Alongside a thyroid ultrasound scan, serum calcitonin is required as part of the workup for MEN type II to look for medullary thyroid cancer. Unlike other thyroid cancers, thyroid function tests are usually normal, and presenting features are usually cervical lymphadenopathy or thyroid lumps.

MRI head is incorrect. MEN type 1 is associated with the '3 Ps' - pituitary tumours (most commonly prolactinomas), pancreatic tumours, and parathyroid tumours. In this case, a prolactinoma is unlikely given the normal LH and FSH (prolactin usually suppresses LH and FSH levels) and there are no signs or symptoms of pancreatic tumours and blood glucose levels are normal. MEN type II is more likely. MRI head imaging to look for pituitary tumours is not indicated.

MRI pancreas is incorrect. In this case, there are no signs or symptoms of pancreatic tumour, and blood glucose is normal. This is, therefore, not the most appropriate option.

Skeletal survey is incorrect. This would form part of the workup for multiple myeloma. Although there is hypercalcemia here, there are no other signs or symptoms of multiple myeloma. A diagnosis of multiple myeloma would not explain the headaches precipitated by activity and caffeine. This is, therefore, not the correct answer.

Ultrasound adrenals is incorrect. Adrenal imaging is important in order to assess for pheochromocytoma. Ultrasound, however, is not the appropriate imaging modality - CT scanning is used.

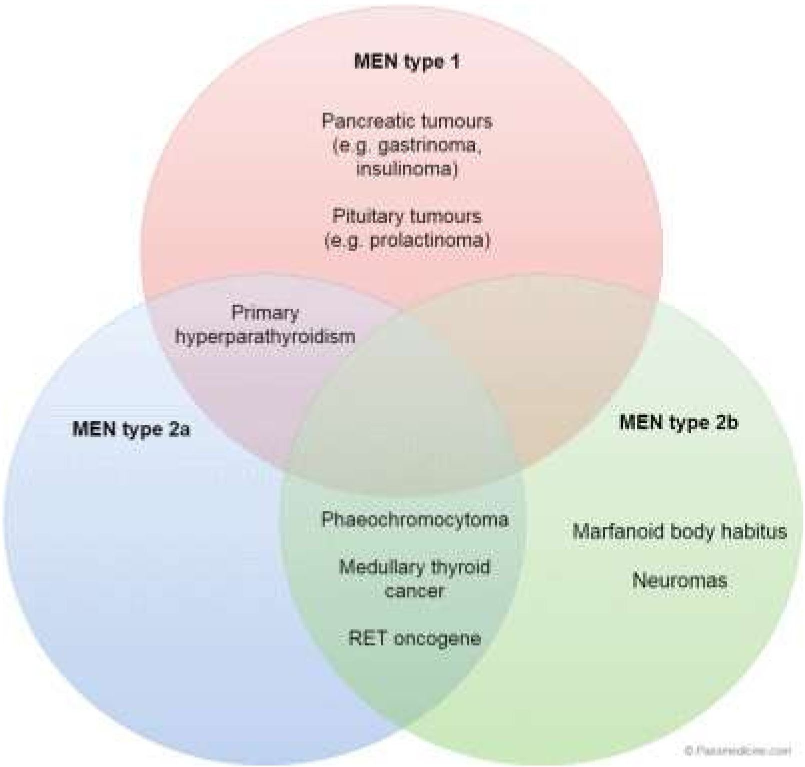
👍 👎 Discuss (5) Improve

Next question

Multiple endocrine neoplasia ★

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
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Venn diagram showing the different types of MEN and their associated features

Q 123
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PANCREATIC
NEUROENDOCRINE
NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

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Score: **66.7%**

1 ✓
2 ✓
3 ✗

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← Question 4 of 101 →

A 27-year-old woman has been referred to the endocrinology clinic. She has been complaining of palpitations over the last three weeks, and an ECG during an episode of palpitation revealed sinus tachycardia. This resolved with a Valsalva manoeuvre. She was offered but declined any beta-blockers. She has no past medical history and does not take any regular medications apart from over the counter beta-blockers and an oral contraceptive pill. She does not smoke but drinks roughly two units of alcohol per week. She has a non-tender goitre on examination. Biochemical investigations reveal an undetectable TSH, free T4 of 46ng/dl and positive thyroid-stimulating hormone receptor antibodies. On noticing the goitre, she is keen to start treatment. After discussion, she opts for carbimazole treatment with a view of inducing remission. On starting carbimazole she is warned that if she experiences a sore throat or any infection she must have blood tests to exclude agranulocytosis. What other symptoms is it important to warn her about?

- ○ Loss of vision ×
- ○ Loss of peripheral sensation ×
- ● ~~Jaundice~~ ×
- ○ Worsening palpitations ×
- ○ Insomnia ×

Submit answer

Dr. Jamil Alrajhi

Reference ranges

Score: **66.7%**

1	✓
2	✓
3	✗
4	-

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Question 4 of 101 ✓

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Loss of vision	10%
Loss of peripheral sensation	19%
Jaundice	47%
Worsening palpitations	9%
Insomnia	14%

Patients taking carbimazole need to be warned about potential hepatic impairment and the relevant symptoms

Important for me Less important

This is a patient with clinical and biochemical evidence of Graves' disease. As this has been confirmed with positive thyroid-stimulating hormone receptor antibodies it would be entirely appropriate to offer carbimazole, radioiodine or surgery. When starting carbimazole, it is very important to ensure the patient is aware of the risk of agranulocytosis, but it is also important to warn about liver dysfunction which may develop and may necessitate stopping the treatment. Rash, headache, fever and malaise commonly occur as well, but can usually be managed with analgesia and antihistamines. Loss of vision could occur in the case of moderate to severe Graves' disease with orbitopathy and starting on radio-iodine, which can worsen thyroid eye disease. Palpitations and insomnia should improve with treatment as these are symptoms of hyperthyroidism.

Discuss (9) Improve

Next question

Carbimazole

Carbimazole is used in the management of thyrotoxicosis. It is typically given in high doses for 6 weeks until the patient becomes euthyroid before being reduced.

Mechanism of action

- blocks thyroid peroxidase from coupling and iodinating the tyrosine residues on thyroglobulin → reducing thyroid hormone production
- in contrast propylthiouracil as well as this central mechanism of action also has a peripheral action by inhibiting 5'-deiodinase which reduces peripheral conversion of T4 to T3

Adverse effects

- agranulocytosis
- crosses the placenta, but may be used in low doses during pregnancy

+ Q 123

Next question

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HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 6 👎 1

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Score: **75%**

1 ✓
2 ✓
3 ✗
4 ✓

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Question 5 of 101

A 23-year-old man is diagnosed as having type 1 diabetes mellitus after presenting with diabetic ketoacidosis. His blood sugars are now stable and he is well. What is the first-line insulin regime he should be offered?

- ○ Twice-daily mixed insulin ×
- ○ Once-daily mixed insulin ×
- ○ Basal-bolus insulin regimen with twice-daily insulin detemir ×
- ○ Basal-bolus insulin regimen with once-daily insulin glargine ×
- ○ Rapid-acting insulin analogue before each meal with no longer acting insulin ×

Submit answer

Reference ranges

Score: **75%**

1	✓
2	✓
3	✗
4	✓
5	-

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← Question 5 of 101 ✗ ⚐ →

A 23-year-old man is diagnosed as having type 1 diabetes mellitus after presenting with diabetic ketoacidosis. His blood sugars are now stable and he is well. What is the first-line insulin regime he should be offered?

	Twice-daily mixed insulin	7%
	Once-daily mixed insulin	1%
🟩	Basal-bolus insulin regimen with twice-daily insulin detemir	32%
🟥	Basal-bolus insulin regimen with once-daily insulin glargine	57%
	Rapid-acting insulin analogue before each meal with no longer acting insulin	3%

In newly diagnosed adults with type 1 diabetes, the first-line insulin regime should be a basal-bolus using twice-daily insulin detemir

Important for me Less important

👍 👎 Discuss (7) Improve

Next question

Diabetes mellitus: management of type 1 ★

The long-term management of type 1 diabetics is an important and complex process requiring the input of many different clinical specialties and members of the healthcare team. A diagnosis of type 1 diabetes can still reduce the life expectancy of patients by 13 years and the micro and macrovascular complications are well documented.

NICE released guidelines on the diagnosis and management of type 1 diabetes in 2015. We've only highlighted a very select amount of the guidance here which will be useful for any clinician looking after a patient with type 1 diabetes.

HbA1c
- should be monitored every 3-6 months
- adults should have a target of HbA1c level of 48 mmol/mol (6.5%) or lower. NICE do however recommend taking into account factors such as the person's daily activities, aspirations, likelihood of complications, comorbidities, occupation and history of hypoglycaemia

Self-monitoring of blood glucose
- recommend testing at least 4 times a day, including before each meal and before bed
- more frequent monitoring is recommended if frequency of hypoglycaemic episodes increases; during periods of illness; before, during and after sport; when planning pregnancy, during pregnancy and while breastfeeding

Blood glucose targets
- 5-7 mmol/l on waking and
- 4-7 mmol/l before meals at other times of the day

Type of insulin
- offer multiple daily injection basal-bolus insulin regimens, rather than twice-daily mixed insulin regimens, as the insulin injection regimen of choice for all adults
- twice-daily insulin detemir is the regime of choice. Once-daily insulin glargine or insulin detemir is an alternative
- offer rapid-acting insulin analogues injected before meals, rather than rapid-acting soluble human or animal insulins, for mealtime insulin replacement for adults with type 1 diabetes

Metformin

- NICE recommend considering adding metformin if the BMI >= 25 kg/m²

+ Q 123

Next question

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Links

NICE 👍 8 👎 6

2015 Diabetes (type 1 and type 2) in children and young people

NICE 👍 15 👎 7

2015 Type 1 diabetes in adults: diagnosis and management

Suggest link Report broken link

Score: **60%**

1 ✓

2 ✓

3 ✗

4 ✓

5 ✗

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A 32-year-old woman presents with recurrent headaches and blurred vision. She is fatigued and has recurrent constipation requiring laxatives. There is a past medical history of hypertension and she is on amlodipine, ramipril and bendroflumethiazide.

On examination, her blood pressure is 178/102 mmHg. Upon fluid balance review, she seems hypervolaemic with bilateral peripheral oedema. She is of slim build and her cranial nerves and neurological examination show no abnormalities. Fundoscopy reveals grade IV hypertensive retinopathy with papilloedema seen.

Her electrocardiogram reveals features of left ventricular hypertrophy. Her urine dip is negative for protein and blood. A CT head reveals no acute bleed.

Blood results are as follows:

Na^+	154 mmol/L	(135 - 145)
K^+	2.9 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Aldosterone	70 pmol/L	(100 - 500)
Renin	2 mU/L	(5-50)

What is the most likely diagnosis?

○ Bartter's syndrome ✕

○ Excess salt in diet ✕

○ Gitelman's syndrome ✕

○ Liddle syndrome ✕

○ Renal artery stenosis ×

Submit answer

Reference ranges

Score: **60%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	-

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Question 6 of 101 ✓

A 32-year-old woman presents with recurrent headaches and blurred vision. She is fatigued and has recurrent constipation requiring laxatives. There is a past medical history of hypertension and she is on amlodipine, ramipril and bendroflumethiazide.

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Bicarbonate	23 mmol/L	(22 - 29)
Aldosterone	70 pmol/L	(100 - 500)
Renin	2 mU/L	(5-50)

What is the most likely diagnosis?

Bartter's syndrome	6%
Excess salt in diet	1%
Gitelman's syndrome	7%
[✓ Liddle syndrome]	73%

Renal artery stenosis 13%

Hypernatraemia associated with hypervolaemia can occur due to hypertonic saline, hypertonic sodium bicarbonate, excess salt in diet, or hyperaldosteronism

Important for me Less important

Liddle syndrome is the correct option. This rare autosomal dominant syndrome is due to dysregulation of the epithelial sodium channel (ENaC) due to a genetic mutation at the 16p13-p12 locus, leading to the Na channel not being degraded, thus leading to chronic reabsorption of sodium and water in the nephron. This mimics a state of hyperaldosteronism as aldosterone is usually the hormone implicated in inserting the ENaCs into the nephron. Therefore, this patient has a state of hypervolaemic hypernatraemia with chronically high blood pressure creating the end-organ damage to the optic nerve and heart described above.

The feedback loop in the kidneys has meant that both renin and aldosterone are low for her body to attempt to lower the sodium and water retention. This patient is fatigued and constipated due to hypokalaemia. Liddle's is treated with a combination of a low sodium diet and potassium-sparing diuretics (e.g. amiloride).

Liddle's has a classic triad in the textbooks of hypertension, hypokalemia and metabolic alkalosis. However, hypernatraemia is also a feature due to the above processes and this should not be forgotten in the clinical work-up.

Bartter's syndrome is an incorrect option. This is an autosomal recessive syndrome which causes salt-wasting from the kidney and subsequently a state of hyperaldosteronism and usually low blood pressure, not high blood pressure as seen in this case.

Gitelman's syndrome is an incorrect option as it is another autosomal recessive syndrome similar to Bartter's, which causes low blood pressure usually.

Excess salt in the diet is incorrect. Even though it would cause hypernatraemia and chronic excess would cause states of hypertension; you would not find hypokalaemia. Furthermore, it would be a more unlikely cause at the young age of this patient with the anti-hypertensive therapy resistance.

Renal artery stenosis is an incorrect answer because this condition causes difficulty to control hypertension with a high plasma renin activity found, instead of low, as more renin is produced in response to the low pressure sensed by the constricted renal artery. The kidneys think that the blood pressure is systemically low when it is a locally mediated effect from the stenosis.

👍 👎 Discuss (6) Improve

Next question

Hypernatraemia

Causes of hypernatraemia
- dehydration
- osmotic diuresis e.g. hyperosmolar non-ketotic diabetic coma
- diabetes insipidus
- excess IV saline

Hypernatraemia should be corrected with great caution. Although brain tissue can lose sodium and potassium rapidly, lowering of other osmolytes (and importantly water) occurs at a slower rate, predisposing to cerebral oedema, resulting in seizures, coma and death1. Although there are no clinical guidelines by NICE or Royal College of Physicians at present, it is generally accepted that a rate of no greater than 0.5 mmol/hour correction is appropriate[1].

1. Reynolds RM, Padfield PL, Seckl JR; Disorders of sodium balance. BMJ. 2006 Mar 25;332(7543):702-5.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **66.7%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓

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Question 7 of 101

A 54-year-old man with a history of type 2 diabetes is recovering on the surgical ward having suffered an episode of acute pancreatitis some 4 days earlier. Medication for glucose control includes metformin, dapagliflozin and liraglutide. On examination his blood pressure is 135/80 mmHg, pulse is 72 and regular. His body mass index is 35 kg/m^2. A recent HbA1c is 63 mmol/mol, renal function is reported as normal. Which of the following is the correct course of action with respect to his long term blood glucose lowering medication?

- ○ Stop metformin ×
- ○ Stop dapagliflozin ×
- ○ Stop liraglutide ×
- ○ Continue usual medication ×
- ○ Stop metformin and liraglutide ×

Submit answer

Reference ranges

Score: **66.7%**

1 ✓
2 ✓
3 ✗
4 ✓

5	✗
6	✓
7	-

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A 54-year-old man with a history of type 2 diabetes is recovering on the surgical ward having suffered an episode of acute pancreatitis some 4 days earlier. Medication for glucose control includes metformin, dapagliflozin and liraglutide. On examination his blood pressure is 135/80 mmHg, pulse is 72 and regular. His body mass index is 35 kg/m². A recent HbA1c is 63 mmol/mol, renal function is reported as normal. Which of the following is the correct course of action with respect to his long term blood glucose lowering medication?

	Stop metformin	3%
	Stop dapagliflozin	13%
🟩	Stop liraglutide	65%
🟥	Continue usual medication	14%
	Stop metformin and liraglutide	4%

This patient is now stable following his episode of pancreatitis, but in view of a number of studies suggesting an association between GLP-1 agonist prescription and pancreatitis, it should be withdrawn in patients who suffer pancreatitis whilst on therapy, and not instituted in patients who have a history of the disease. For this reason liraglutide should be withdrawn.

With respect to metformin, it should potentially be discontinued during periods of increased risk of tissue hypoxia, (e.g. during the acute period of pancreatitis), but there is no indication it should be omitted over the longer term. In regards to metformin and renal impairment, guidance has recently been revised to support metformin initiation at glomerular filtration rate as low as 45ml/min.

👍 👎 Discuss (3) Improve

Next question

Diabetes mellitus: management of type 2

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

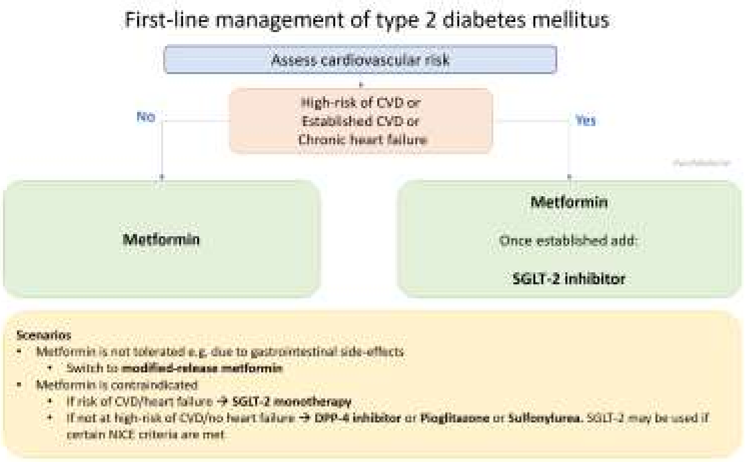
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
    - Switch to **modified-release metformin**
- Metformin is contraindicated
    - If risk of CVD/heart failure → **SGLT-2 monotherapy**
    - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

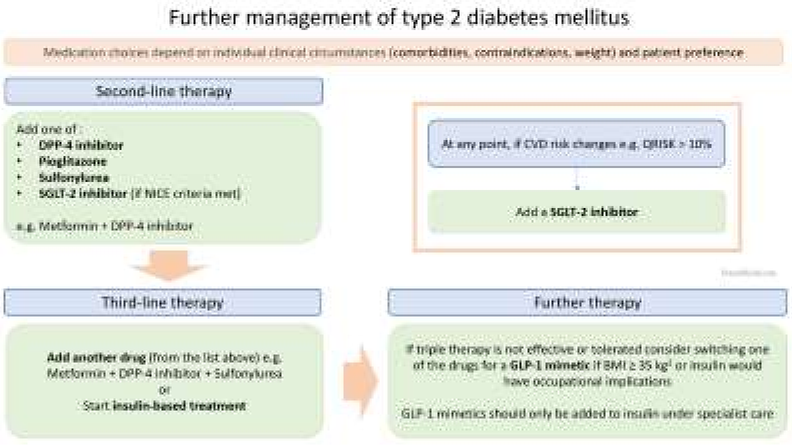
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:

    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

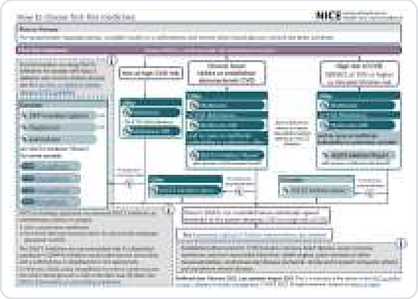
Starting insulin

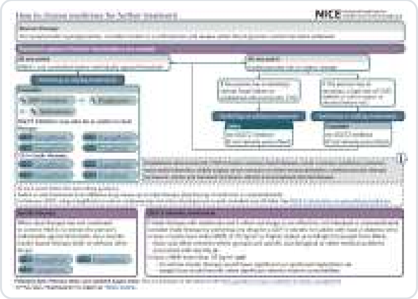
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

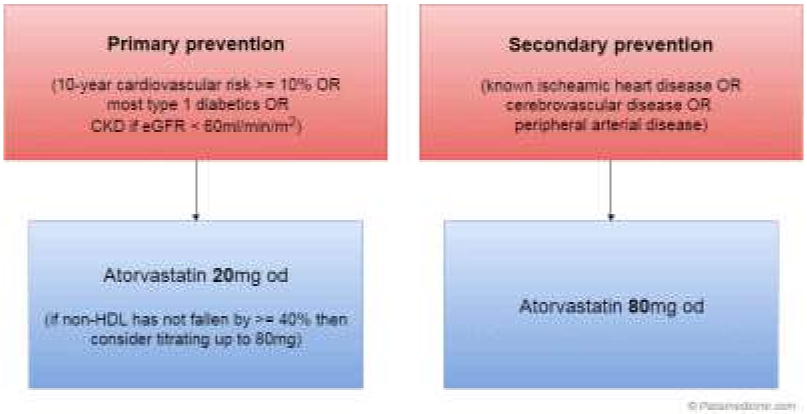
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

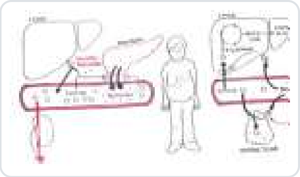
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

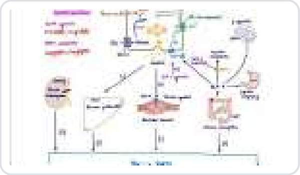
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **57.1%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓
7 ✗

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A 53-year-old woman comes for review in the general medical clinic. She was diagnosed with type 2 diabetes mellitus six months ago after having developed fatigue and polyuria. She also has hypothyroidism but no other comorbidities. She was started on metformin 500mg twice daily struggled to cope due to gastrointestinal side effects such as diarrhoea. What is the most appropriate action?

- ○ Reduce to metformin 500mg once daily ×
- ○ Change to dipeptidyl peptidase-4 inhibitor ×
- ○ Trial of modified release metformin ×
- ○ Change to sulfonylurea ×
- ○ Change to pioglitazone ×

Submit answer

Reference ranges ∨

Score: **57.1%**

1 ✓
2 ✓
3 ✗
4 ✓

5	✗
6	✓
7	✗
8	-

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A 53-year-old woman comes for review in the general medical clinic. She was diagnosed with type 2 diabetes mellitus six months ago after having developed fatigue and polyuria. She also has hypothyroidism but no other comorbidities. She was started on metformin 500mg twice daily struggled to cope due to gastrointestinal side effects such as diarrhoea. What is the most appropriate action?

Reduce to metformin 500mg once daily	3%
Change to dipeptidyl peptidase-4 inhibitor	3%
Trial of modified release metformin	91%
Change to sulfonylurea	2%
Change to pioglitazone	1%

The correct answer trial of modified release metformin. NICE guidelines advise to offer standard release metformin as the first-line treatment for type 2 diabetes and to gradually increase the dose to minimise the risk of gastrointestinal side effects. If gastrointestinal side effects are not tolerated, then a trial of modified release metformin would be appropriate. If metformin is not tolerated at all then a dipeptidyl peptidase-4 inhibitor, sulfonylurea or pioglitazone would be indicated.

Source:

'Type 2 diabetes in adults: management.' NICE guideline [NG28]. The National Institute for Health and Care Excellence, December 2015.

Discuss (1) | Improve

Next question

Diabetes mellitus: management of type 2

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation

- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

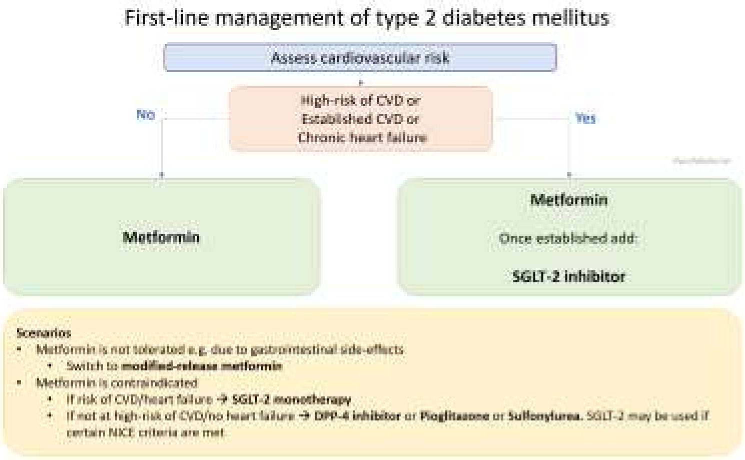
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

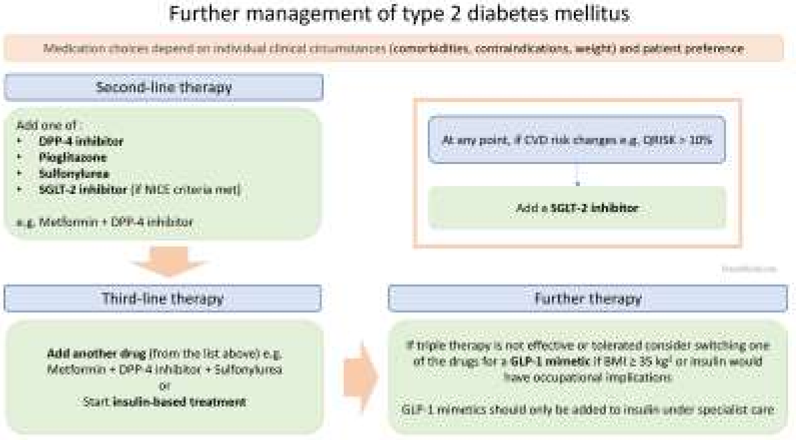
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:

  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea

- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

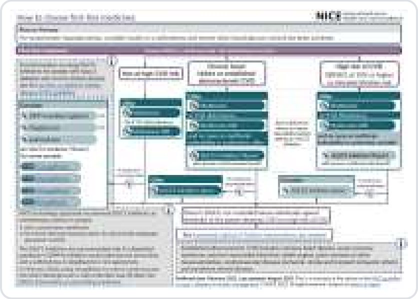
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

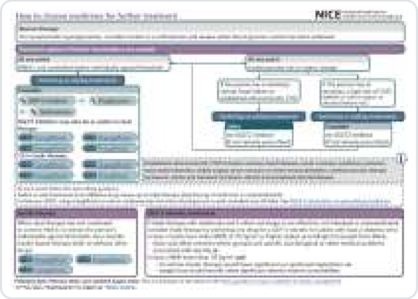
Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples
- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea

Type 2 diabetes in adults: choosing medicines



How to choose first-line medicines



How to choose medicines for further treatment

Summary of first-line medicines

Summary of medicines for further treatment

Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line

    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
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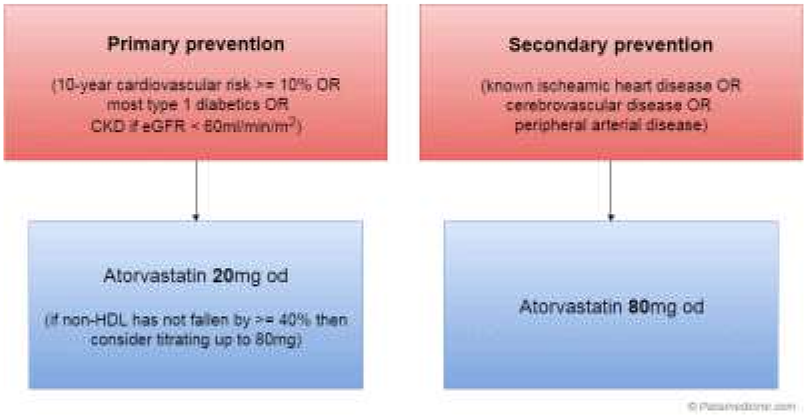
Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
© Passmedicine.com

Graphic showing choice of statin.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

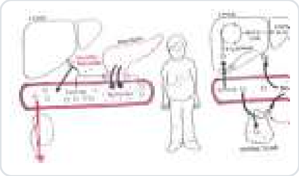
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

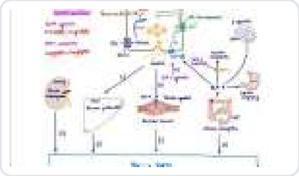
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **62.5%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓

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Question 9 of 101

A 45-year-old woman comes to the clinic some 6 months after thyroid resection for differentiated thyroid cancer. She is well, has recovered from her surgery and has a neatly healed scar across her anterior neck. Blood pressure is normal at 110/80 mmHg, and her pulse is 60 and regular. Her body mass index is unchanged at 25 kg/m². Only medication is thyroid hormone replacement.

Which of the following is the most appropriate way to monitor for a recurrence?

- ○ MRI neck ✕
- ○ Technetium scanning ✕
- ○ Thyroglobulin ✕
- ○ Thyroid ultrasound scan ✕
- ○ T3 levels ✕

Submit answer

Reference ranges

Score: **62.5%**

1 ✓
2 ✓
3 ✗

4	✓
5	✗
6	✓
7	✗
8	✓
9	-

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A 45-year-old woman comes to the clinic some 6 months after thyroid resection for differentiated thyroid cancer. She is well, has recovered from her surgery and has a neatly healed scar across her anterior neck. Blood pressure is normal at 110/80 mmHg, and her pulse is 60 and regular. Her body mass index is unchanged at 25 kg/m². Only medication is thyroid hormone replacement.

Which of the following is the most appropriate way to monitor for a recurrence?

MRI neck	1%
Technetium scanning	3%
Thyroglobulin	90%
Thyroid ultrasound scan	3%
T3 levels	3%

Given this patient is prescribed thyroid hormone replacement, monitoring of T3 or T4 is not useful in monitoring for cancer recurrence. On the other hand the presence of thyroglobulin does indicate thyroid gland activity which isn't suppressed by thyroid hormone replacement and is therefore potentially cancerous in origin. Levels should be undetectable.

Radiological investigations such as MRI neck, technetium scanning or thyroid ultrasound scanning are potentially less sensitive than monitoring for thyroglobulin and are therefore not a preferred first line investigation. T3 levels are unsuitable as a marker of recurrence given that conversion of exogenously administered T4 to T3 confounds any measurement.

👍 👎 Discuss (5) Improve

Next question

Thyroid cancer ★

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

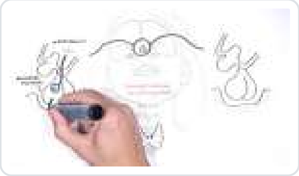
Suggest link Report broken link

Media

THYROID
CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0



Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **66.7%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓

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A 58-year-old man is referred to the endocrinology clinic with a 6-month history of fatigue, reduced libido and occasional headaches. He has noticed some difficulty with peripheral vision over the past few weeks. There is no significant past medical history. On examination, his blood pressure is 108/70 mmHg and he appears pale. Confrontation testing reveals reduced vision in both temporal fields. Laboratory investigations are as follows:

Na^+	133 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Cortisol (9am)	110 nmol/L	(140 - 700)
TSH	1.1 mU/L	(0.4 - 4.0)
Free T4	9 pmol/L	(10 - 22)
LH	<1 IU/L	(Men: 1-8)
FSH	<1 IU/L	(Men: 1-12)
Prolactin	410 mU/L	(<350)

Which of the following is the most appropriate next step in management?

○ Transsphenoidal surgery ×

○ Cabergoline therapy ×

○ Hydrocortisone replacement only ×

○ Pituitary radiotherapy ×

○ Repeat pituitary hormone profile in three months ×

Submit answer

Reference ranges

Score: **66.7%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	-

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Question 10 of 101 ✗

A 58-year-old man is referred to the endocrinology clinic with a 6-month history of fatigue, reduced libido and occasional headaches. He has noticed some difficulty with peripheral vision over the past few weeks. There is no significant past medical history. On examination, his blood pressure is 108/70 mmHg and he appears pale. Confrontation testing reveals reduced vision in both temporal fields. Laboratory investigations are as follows:

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Free T4	9 pmol/L	(10 - 22)
LH	<1 IU/L	(Men: 1-8)
FSH	<1 IU/L	(Men: 1-12)
Prolactin	410 mU/L	(<350)

Which of the following is the most appropriate next step in management?

🟩	Transsphenoidal surgery	69%
	Cabergoline therapy	23%
	Hydrocortisone replacement only	5%
	Pituitary radiotherapy	1%
🟥	Repeat pituitary hormone profile in three months	1%

Non-functioning pituitary tumours present with hypopituitarism and pressure effects

Important for me Less important

Transsphenoidal surgery is the correct answer because this patient has clinical and biochemical evidence of hypopituitarism—fatigue, low libido, hypotension, hyponatraemia, secondary hypothyroidism and hypogonadism—and signs of mass effect from a pituitary lesion such as bitemporal hemianopia and headache. The modestly elevated prolactin level is consistent with stalk effect rather than a prolactinoma, as levels are typically much higher (>2000 mU/L) in true prolactinomas. UK guidelines recommend urgent surgical decompression for non-functioning pituitary macroadenomas presenting with visual field defects or significant mass effect to relieve compression on the optic chiasm and restore normal pituitary function where possible.

Cabergoline therapy would be first-line for a confirmed prolactinoma; however, this patient's mildly raised prolactin likely reflects stalk compression ('disconnection hyperprolactinaemia') by a non-functioning tumour rather than autonomous secretion by a prolactinoma, which would present with much higher levels and typically without panhypopituitarism or visual loss.

Hydrocortisone replacement only addresses one aspect of his adrenal insufficiency but does not address the underlying cause—the compressive pituitary mass causing multiple hormone deficiencies and visual compromise—which requires surgical intervention.

Pituitary radiotherapy can be considered for residual or recurrent tumours post-surgery or when surgery is contraindicated or unsuccessful at controlling tumour growth; it is not used as first-line treatment for symptomatic non-functioning macroadenomas causing acute visual symptoms.

Repeat pituitary hormone profile in three months would delay appropriate management in a patient with clear evidence of hypopituitarism and progressive neurological deficit due to mass effect; prompt intervention to prevent further visual loss and treat hormonal insufficiency is required.

👍 👎 Discuss (1) Improve

Next question

Pituitary adenoma

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:
- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due to excess prolactin)
- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
  - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
  - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).
  - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
  - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone) and neuromodulators like pasireotide
- transsphenoidal surgery
  - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
  - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
  - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
  - indicated for residual or recurrent tumours post-surgery

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 20 👎 11

Pituitary tumours review

Suggest link Report broken link

Score: **60%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓

7	✗
8	✓
9	✓
10	✗

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A 45-year-old man presents to review to the endocrinology clinic. He has been suffering from erectile dysfunction for six months and seen his GP for this. He has had serum testosterone tested and this was found to be low. This is confirmed on repeat testing. LH and FSH are found to be within normal range. The patient describes erectile dysfunction as well as loss of nocturnal erections. He describes increased stress at work due to a recent promotion. He also feels that the erectile dysfunction has put a strain on his marriage. He also has been suffering from worsening headaches, occurring at night, loss of energy and loss of sexual desire.

What is the most likely aetiology for his erectile dysfunction?

- ○ Psychological ×
- ○ Acquired primary hypogonadism ×
- ○ Genetic primary hypogonadism ×
- ○ Secondary hypogonadism ×
- ○ Androgen insensitivity syndrome ×

Submit answer

Reference ranges

Score: **60%**

1 ✓

2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✗
11	-

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What is the most likely aetiology for his erectile dysfunction?

	Psychological	26%
🟥	Acquired primary hypogonadism	23%
	Genetic primary hypogonadism	2%
🟩	Secondary hypogonadism	46%
	Androgen insensitivity syndrome	3%

In male patients with low libido have been found to have a low testosterone first line investigation should include prolactin and LH to assess for a central cause

Important for me Less important

The patient describes symptoms of hypogonadism with loss of sexual desire, erectile dysfunction and low energy. This is supported by low morning testosterone. This combination makes a psychological cause unlikely. As the LH and FSH are not elevated a primary hypogonadism is excluded. In either acquired or genetic primary hypogonadism there would be increased LH and FSH due to loss of negative feedback. In secondary hypogonadism, LH and FSH are low or normal with low testosterone. Potential cause include increased prolactin, which can be by tumour or drug-induced. Prolactin reduces LH and FSH and can therefore be a cause. Other potential secondary causes of hypogonadism include hypopituitarism, Kallmann's syndrome and isolated hypogonadotrophic hypogonadism. Androgen insensitivity syndrome is associated with elevated testosterone.

👍 👎 Discuss (14) Improve

Next question

Disorders of sex hormones

The table below summarises the findings in patients who have disorders of sex hormones:

Disorder	LH	Testosterone
Primary hypogonadism (Klinefelter's syndrome)	High	Low
Hypogonadotrophic hypogonadism (Kallman's syndrome)	Low	Low
Androgen insensitivity syndrome	High	Normal/high
Testosterone-secreting tumour	Low	High

Klinefelter's syndrome

Klinefelter's syndrome is associated with karyotype 47, XXY

Features
- often taller than average
- lack of secondary sexual characteristics
- small, firm testes

- infertile
- gynaecomastia - increased incidence of breast cancer
- elevated gonadotrophin levels

Diagnosis is by chromosomal analysis

Kallman's syndrome

Kallman's syndrome is a recognised cause of delayed puberty secondary to hypogonadotrophic hypogonadism. It is usually inherited as an X-linked recessive trait. Kallman's syndrome is thought to be caused by failure of GnRH-secreting neurons to migrate to the hypothalamus.

The clue given in many questions is lack of smell (anosmia) in a boy with delayed puberty

Features
- 'delayed puberty'
- hypogonadism, cryptorchidism
- anosmia
- sex hormone levels are low
- LH, FSH levels are inappropriately low/normal
- patients are typically of normal or above average height

Cleft lip/palate and visual/hearing defects are also seen in some patients

Androgen insensitivity syndrome

Androgen insensitivity syndrome is an X-linked recessive condition due to end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Complete androgen insensitivity syndrome is the new term for testicular feminisation syndrome

Features

- 'primary amenorrhoea'
- undescended testes causing groin swellings
- breast development may occur as a result of conversion of testosterone to oestradiol

Diagnosis

- buccal smear or chromosomal analysis to reveal 46XY genotype

Management

- counselling - raise child as female
- bilateral orchidectomy (increased risk of testicular cancer due to undescended testes)
- oestrogen therapy

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Dr. Jamil Alrajhi

Score: **54.5%**

1 ✓

2 ✓

3 ✗

4 ✓

5 ✗

6 ✓

7 ✗

8 ✓

9 ✓

10 ✗

11 ✗

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← Question 12 of 101 →

A 24-year-old female presents with one week of progressive and persistent double vision. She reports increasing tiredness at all times of day over the past 2 months and occasional chest tightness associated with palpitations. She has no past medical history. She was also adopted and unaware of any family history. On examination, you find a loss of left eye abduction, right eye upwards gaze, right eye adduction. Systemic examination also reveals bilateral clammy hands and a heart rate of 120 per minute, irregular. Which test is most likely to be diagnostic?

- ○ Autoimmune screen ✕
- ○ Thyroid function tests ✕
- ○ CT thorax ✕
- ○ Anti-acetylcholine receptor antibodies ✕
- ○ 12 lead ECG ✕

Submit answer

Reference ranges

Score: **54.5%**

1 ✓
2 ✓
3 ✗
4 ✓

5	✗
6	✓
7	✗
8	✓
9	✓
10	✗
11	✗
12	-

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	Autoimmune screen	8%
🟩	Thyroid function tests	68%
	CT thorax	8%
	Anti-acetylcholine receptor antibodies	15%
🟥	12 lead ECG	1%

This patient presents with systemic symptoms and a complex ophthalmoplegia, the diagnosis of thyroid eye disease, secondary to Graves disease, is most likely. The important test would be thyroid function tests and also MRI of her orbits, which would almost certainly demonstrate retro-orbital and extraocular muscle inflammation. The severity of the patient's eye disease needs to be assessed: the most frequently used criteria was developed by the American thyroid association, which spells out helpfully NO SPECS

Class 0 No symptoms or signs
Class I Only signs, no symptoms (lid retraction, stare, lid lag)
Class II Soft tissue involvement
Class III Proptosis
Class IV Extraocular muscle involvement
Class V Corneal involvement

Class VI Sight loss (optic nerve involvement)

Any patient presenting with eye movement weaknesses that cannot be explained by isolated or multiple cranial nerve palsies is called complex ophthalmoplegia. The differentials include myasthenia gravis, mononeuritis multiplex, thyroid eye disease, Kearns-Sayre syndrome, complex progressive external ophthalmoplegia, Miller-Fisher syndrome and botulinum poisoning.

👍 👎 Discuss (8) Improve

Next question

Thyroid eye disease

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology

- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention

- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features

- the patient may be eu-, hypo- or hyperthyroid at the time of presentation
- exophthalmos
- conjunctival oedema

- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management
- smoking cessation
- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery

Complications

Exposure keratopathy
- this is the most common complication of thyroid eye disease
- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy
- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia
- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)
- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):

- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes
- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

+ Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy

Management of Graves' Orbitopathy

👍 9 👎 8

Suggest link

Report broken link

Score: **50%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
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9	✓
10	✗
11	✗
12	✗

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You review a 68-year-old patient in the diabetic clinic. He was diagnosed 28 years ago with type 2 diabetes and over this time has been through a number of antiglycemic agents including biguanides, sulfonylureas, thiazolidinediones and insulin. He is generally well but reports painless macroscopic haematuria and would like to be referred to a urologist as he has read about bladder cancer associated with one of his medications.

Which of the following antiglycemic agents can cause bladder cancer?

- ○ Gliclazide ×
- ○ Tolbutamide ×
- ○ Pioglitazone ×
- ○ Insulin detemir ×
- ○ Sitagliptin ×

Submit answer

Reference ranges

Score: **50%**

1	✓
2	✓
3	✗

4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✗
11	✗
12	✗
13	-

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Question 13 of 101 ✓

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Which of the following antiglycemic agents can cause bladder cancer?

Gliclazide	1%
Tolbutamide	4%
Pioglitazone	93%
Insulin detemir	1%
Sitagliptin	1%

Thiazolidinediones are associated with an increased risk of bladder cancer

Important for me Less important

Answer: Pioglitazone

Pioglitazone has been associated with an increased risk of bladder cancer. The greatest risk was shown in those patients who have used pioglitazone long term.

Risk of Bladder Cancer Among Diabetic Patients Treated With Pioglitazone Interim report of a longitudinal cohort study
http://care.diabetesjournals.org/content/34/4/916

Discuss (5) Improve

Next question

Thiazolidinediones

Thiazolidinediones are a class of agents used in the treatment of type 2 diabetes mellitus. They are agonists to the PPAR-gamma receptor and reduce peripheral insulin resistance. Rosiglitazone was withdrawn in 2010 following concerns about the cardiovascular side-effect profile.

The PPAR-gamma receptor is an intracellular nuclear receptor. It's natural ligands are free fatty acids and it is thought to control adipocyte differentiation and function.

Adverse effects

- weight gain
- liver impairment: monitor LFTs
- fluid retention - therefore contraindicated in heart failure. The risk of fluid retention is increased if the patient also takes insulin
- recent studies have indicated an increased risk of fractures
- bladder cancer: recent studies have shown an increased risk of bladder cancer in patients taking pioglitazone (hazard ratio 2.64)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 11 👎 7

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Score: **53.8%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✗
11	✗
12	✗
13	✓

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← Question 14 of 101 →

A 50-year-old gentleman presents to review in the endocrine clinic. He was normally taking metformin and gliclazide, but over the last six months has also been prescribed a GLP-1 mimetic. He was started on a GLP-1 mimetic because of poor diabetic control and concerns about starting insulin. He does not want to start insulin because he is a truck driver and worried about losing his driving license. He had previously tried the combination of metformin, gliclazide and pioglitazone, but this had also failed to control his HbA1c.

Over the last six months his HbA1c has reduced from 81mmol/ mol to 80mmol/ mol, and he has lost two kg in weight. What is the most appropriate action?

○ Continue current treatment and review in one year ×

○ Continue current treatment and review in six months ×

○ Change to metformin, gliclazide and pioglitazone ×

○ Offer to add on insulin ×

○ Stop GLP-1 mimetic and offer insulin treatment ×

Submit answer

Reference ranges

Score: **53.8%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
10	✗
11	✗
12	✗
13	✓
14	-

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Over the last six months his HbA1c has reduced from 81mmol/ mol to 80mmol/ mol, and he has lost two kg in weight. What is the most appropriate action?

Continue current treatment and review in one year	2%
Continue current treatment and review in six months	27%
Change to metformin, gliclazide and pioglitazone	2%
Offer to add on insulin	17%
Stop GLP-1 mimetic and offer insulin treatment	51%

The correct answer is to stop GLP-1 mimetic and offer insulin treatment. This is a patient with diabetes whose diabetic control has not improved even when on a combination of metformin and two other drugs. He was then started on GLP-1 mimetic therapy, but nice advice that this should only be continued if after six months HbA1c reduces by at least 11 mmol/mol, which has not happened. Therefore continuing the current treatment is incorrect. Insulin should not be combined with a GLP-1 mimetic.

Source:

'Type 2 diabetes in adults: management.' NICE guideline [NG28]. The National Institute for Health and Care Excellence, December 2015.

Discuss (13) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

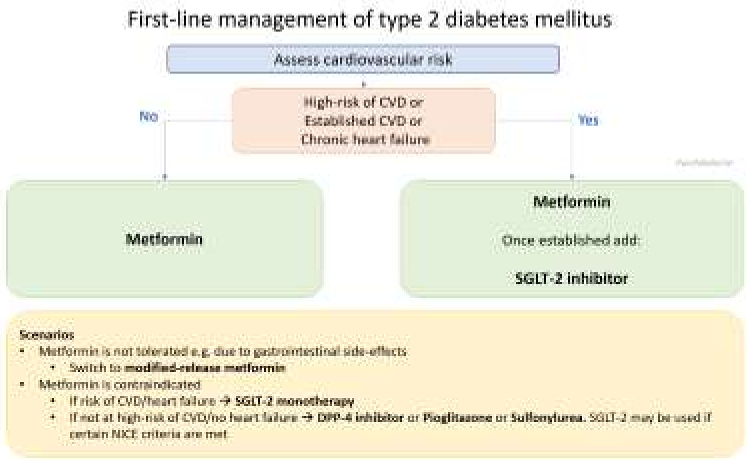
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

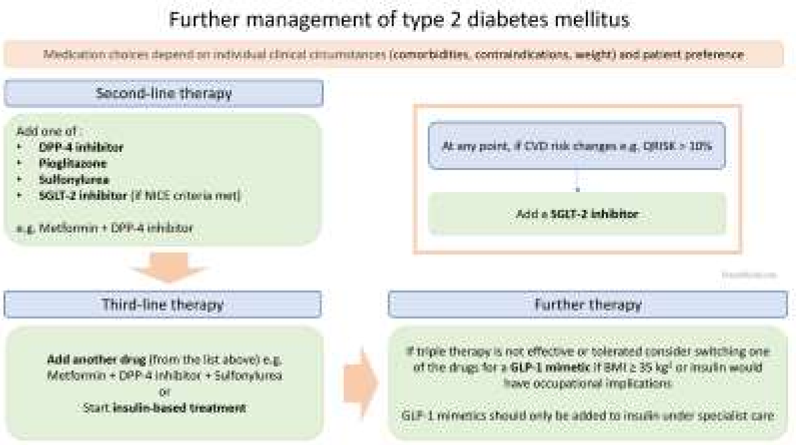
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

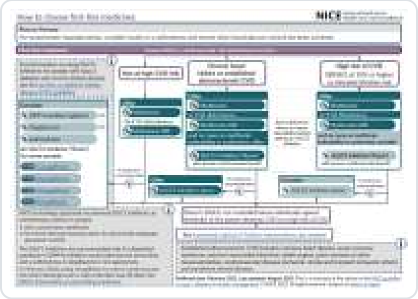
Starting insulin

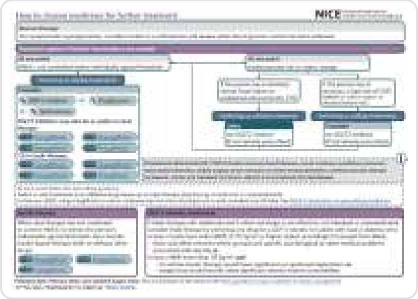
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

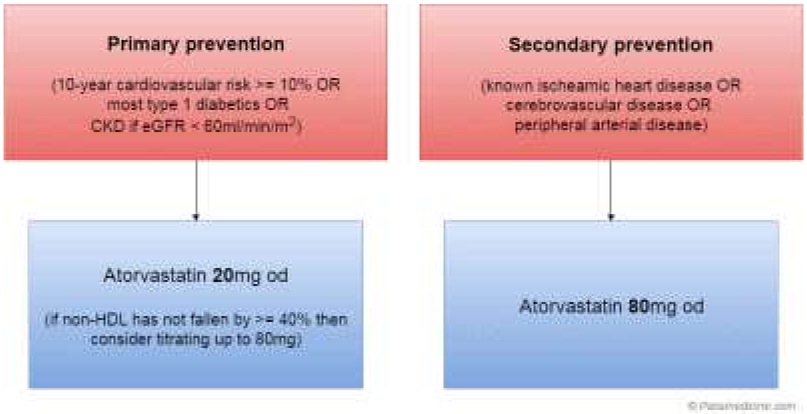
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

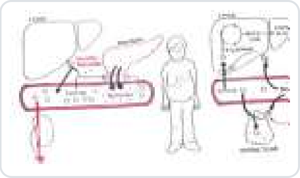
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

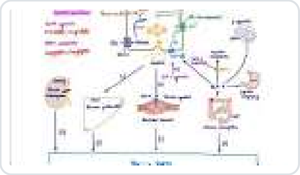
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **57.1%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
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8	✓

9	✓
10	✗
11	✗
12	✗
13	✓
14	✓

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A 19-year-old man is brought by ambulance to the emergency department. The patient himself was too unwell to provide a coherent history but his mother reports that he had been very unwell with loss of appetite and abdominal pain for the past two days. She had also noted a significant weight loss in her son over the past six months and that he had been unusually fatigued over the same time period. The patient had no previous family history although his older sister had recently been diagnosed with pernicious anaemia.

Examination showed the patient to be tachypnoeic and tachycardic with a central capillary refill time of six seconds. Respiratory and cardiovascular examination was otherwise unremarkable. Abdominal examination revealed some inconsistent tenderness without obvious signs of localised peritonism.

Please see below for results of initial investigations.

Urea	10.6 mmol / L
Creatinine	134 micromol / L
Sodium	149 mmol / L
Potassium	5.8 mmol / L
Fingerpick blood glucose	42 mmol / L
Fingerpick blood ketones	7.5 mmol / L

Venous blood gas (room air)

pH	7.09
PCO2	16 mmHg (reference 32-43)
PO2	52 mmHg (reference 70-100)
Bicarbonate	10.9 mmol / L (reference 20.0-26.0)
Chloride	107 mmol / L (reference 99-108)
Lactate	4.9 mmol / L

Portable chest x-ray: poor quality film secondary overlying artefact; no free air under diaphragm, lung fields clear, no pneumothorax.

Electrocardiogram: sinus tachycardia at 129; normal intervals and axis; QRS morphology normal; T wave morphology normal.

What is the correct immediate management for this patient?

- ○ 1000 mg calcium gluconate ✕
- ○ 10 mL 8.4 % sodium bicarbonate ✕
- ● 1000 mL 0.9 % saline ✕
- ○ 1000 ml 0.45 % saline ✕
- ○ Intravenous insulin bolus ✕

Submit answer

Reference ranges

Score: **57.1%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓

9	✓
10	✗
11	✗
12	✗
13	✓
14	✓
15	-

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Lactate	4.9 mmol / L

Portable chest x-ray: poor quality film secondary overlying artefact; no free air under diaphragm, lung fields clear, no pneumothorax.

Electrocardiogram: sinus tachycardia at 129; normal intervals and axis; QRS morphology normal; T wave morphology normal.

What is the correct immediate management for this patient?

1000 mg calcium gluconate	3%
10 mL 8.4 % sodium bicarbonate	2%
[✓ 1000 mL 0.9 % saline]	91%
1000 ml 0.45 % saline	2%
Intravenous insulin bolus	2%

The patient has clear biochemical evidence of diabetic keto-acidosis, most likely representing a first presentation of type 1 diabetes mellitus.

Correct immediate management is to give a litre of 0.9 % saline to restore intravascular volume while preventing a rapid change in extracellular osmolality. For subsequent IV fluids, The Joint British Diabetes Society recommends ongoing use of 0.9 % saline, in contrast to the American Diabetes Association that recommends 0.45 % saline if sodium level normal or high.

An initial bolus dose of intravenous insulin is not recommended after a randomisation controlled trial evidence found no benefit. A weight-based insulin infusion of 0.1 units/kg/hour is recommended.

Bicarbonate infusion are not routinely recommended in the treatment of DKA, although may be considered in patients with severe acidosis (pH < 7) and associated life-threatening effects of acidaemia.

Given the absence of ECG changes of hyperkalaemia in this patient, an infusion of calcium gluconate is not required. In fact, the patient is likely to be more at risk of hypokalaemia as the treatment for DKA progresses.

Misra S, Oliver N. Diabetic ketoacidosis in adults. BMJ 2015;351:h5660.

👍 | 👎 | Discuss (3) | Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points - glucose > 13.8 mmol/l - pH < 7.30 - serum bicarbonate <18 mmol/l - anion gap > 10 - ketonaemia	Key points - glucose > 11 mmol/l or known diabetes mellitus - pH < 7.3 - bicarbonate < 15 mmol/l - ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids
  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and

- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

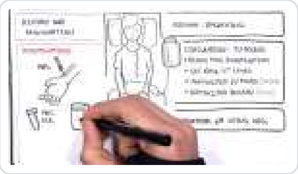
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

Report broken link

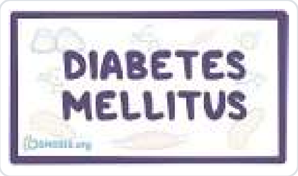
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **60%**

1	✓
2	✓
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14	✓
15	✓

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A 45-year-old lorry driver was referred to the endocrine clinic with symptoms of reduced libido and lack of energy. He had history of a traumatic head injury 5 years previously, which needed a period of 24 hours observation in hospital. On examination, his body mass index was 42 kg/m^2 with normal general physical and systemic examination.

Investigations:

FT4	8.1pmol/l (11.5-22.7)
TSH	0.4mU/l (0.35-5.5)
FSH	2.2U/l (1.4-18.1)
LH	3.5U/l (3.0-8.0)
Testosterone	6.8nmol/l (8.4-28.7)
IGF-1	35nmol/l (16-118)
Prolactin	880mU/l (45-375)

Which one of the following is the most likely diagnosis, based on his clinical profile?

○ Microprolactinoma ×

○ Morbid obesity ×

○ Non-functioning pituitary adenoma ×

○ Post traumatic pituitary apoplexy ×

○ Primary hypothyroidism ×

Submit answer

Reference ranges

Score: **60%**

1	✓
2	✓
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12	✗
13	✓
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← Question 16 of 101 ✗ ⚐ →

A 45-year-old lorry driver was referred to the endocrine clinic with symptoms of reduced libido and lack of energy. He had history of a traumatic head injury 5 years previously, which needed a period of 24 hours observation in hospital. On examination, his body mass index was 42 kg/m^2 with normal general physical and systemic examination.

Investigations:

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Testosterone	6.8nmol/l (8.4-28.7)
IGF-1	35nmol/l (16-118)
Prolactin	880mU/l (45-375)

Which one of the following is the most likely diagnosis, based on his clinical profile?

	Microprolactinoma	29%
🟥	Morbid obesity	11%
🟩	Non-functioning pituitary adenoma	35%
	Post traumatic pituitary apoplexy	20%
	Primary hypothyroidism	5%

The presence of an elevated prolactin level along with secondary hypothyroidism and hypogonadism is indicative of stalk compression is consistent with a non-functioning pituitary adenoma

Important for me Less important

This gentleman has secondary hypothyroidism and hypogonadism in the presence of elevated prolactin levels, which are indicative of stalk compression due to a pituitary adenoma. A prolactinoma is generally associated with prolactin levels >2000 mU/L. Morbid obesity may be associated with hypogonadotrophic hypogonadism, although secondary hypothyroidism is unlikely. Pituitary apoplexy is characterized by a sudden onset of headache, visual symptoms, altered mental status, and hormonal dysfunction due to acute hemorrhage or infarction of a pituitary gland. It usually presents with biochemical features of hypopituitarism which is not presented in this scenario. Primary hypothyroidism is characterized by low FT4, low FT3 and high TSH.

👍 👎 Discuss (10) Improve

Next question

Pituitary adenoma ★

A pituitary adenoma is a benign tumour of the pituitary gland. They are common (10% of all people) but in most cases will never be found (asymptomatic) or are found as an incidental findings. They account for around 10% of adult brain tumours.

Pituitary adenomas can be classified according to:

- **size** (a microadenoma is <1cm and a macroadenoma is ≥1cm)
- **hormonal status** (a secretory/functioning adenoma produces an excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin. After prolactinomas, non-secreting adenomas are the next most common, then GH-secreting and then ACTH-secreting adenomas.

Pituitary adenomas typically cause symptoms by:

- excess of a hormone (e.g. Cushing's disease due to excess ACTH, acromegaly due to excess GH or amenorrhea and galactorrhea due to excess prolactin)
- depletion of a hormone(s) (due to compression of the normal functioning pituitary gland)
  - non-functioning tumours, therefore, present with generalised hypopituitarism
- stretching of the dura within/around the pituitary fossa (causing headaches)
- compression of the optic chiasm (causing a bitemporal hemianopia due to crossing nasal fibers)

Alternatively, pituitary adenomas, particularly microadenomas, can be an incidental finding on neuroimaging and therefore called a 'pituitary incidentaloma'.

Investigation requires:

- a pituitary blood profile (including GH, prolactin, ACTH, FSH, LSH and TFTs)
- formal visual field testing
- MRI brain with contrast

Differential diagnoses include:

- pituitary hyperplasia
- craniopharyngioma
- meningioma
- brain metastases
- lymphoma
- hypophysitis
- vascular malformation (e.g. aneurysm)

Treatment may include a combination of

- medical therapy:
  - prolactinomas are generally treated first-line with dopamine agonists (e.g., cabergoline or bromocriptine).

  - somatostatin analogues (e.g., octreotide, lanreotide) and GH receptor antagonists (e.g., pegvisomant) are used for GH-secreting adenomas
  - for ACTH-secreting adenomas, medical therapy may include cortisol synthesis inhibitors (e.g., ketoconazole, metyrapone) and neuromodulators like pasireotide
- transsphenoidal surgery
  - the primary treatment for most pituitary adenomas is transsphenoidal surgery, especially for non-functioning and ACTH- or GH-secreting adenomas
  - surgical intervention aims to remove the tumour mass, normalise hormone levels, and
  - non-functioning adenomas are generally diagnosed due to their compressive symptoms (e.g. visual problems) or hormone deficiencies - surgery is therefore the first-line treatment
- radiotherapy
  - indicated for residual or recurrent tumours post-surgery

AI + Q 123

Next question

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info

👍 20 👎 11

Pituitary tumours review

Suggest link Report broken link

Score: **56.3%**

1	✓
2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
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12	✗
13	✓
14	✓
15	✓
16	✗

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Question 17 of 101

You are reviewing a 57 year-old gentleman in the diabetes outpatient clinic. He has type 2 diabetes mellitus and is currently taking metformin 850mg three times a day and gliclazide 80mg once daily.

On further questioning he admits having frequent hypoglycaemic episodes at night that distress him as he lives alone. His BMI is calculated at 30.3 kg/m², HbA1c 7.8% (62 mmol/mol) and his co-morbidities include congestive cardiac failure.

How would you change his diabetic treatment?

- ○ Stop gliclazide, start insulin ✕
- ○ Add exenatide ✕
- ○ Add sitagliptin to current regimen ✕
- ○ Stop gliclazide, start pioglitazone ✕
- ○ Stop gliclazide, start sitagliptin ✕

Submit answer

Reference ranges

Score: **56.3%**

1 ✓

2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
9	✓
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12	✗
13	✓
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15	✓
16	✗
17	-

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Question 17 of 101 ✓

You are reviewing a 57 year-old gentleman in the diabetes outpatient clinic. He has type 2 diabetes mellitus and is currently taking metformin 850mg three times a day and gliclazide 80mg once daily.

On further questioning he admits having frequent hypoglycaemic episodes at night that distress him as he lives alone. His BMI is calculated at 30.3 kg/m², HbA1c 7.8% (62 mmol/mol) and his co-morbidities include congestive cardiac failure.

How would you change his diabetic treatment?

	Stop gliclazide, start insulin	3%
	Add exenatide	6%
	Add sitagliptin to current regimen	4%
	Stop gliclazide, start pioglitazone	6%
🟩	Stop gliclazide, start sitagliptin	81%

The NICE guidance on the management of type 2 diabetes mellitus:

- This gentleman has been started on metformin and a sulphonyurea as first line therapy.
- He is having frequent hypoglycaemic episodes secondary to his sulphonylurea and yet control remains poor, HbA1c 7.8% (62 mmol/mol)
- Pioglitazone is contraindicated due to his congestive cardiac failure.
- A DPP-4 inhibitor such as sitagliptin would be a sensible option, the sulphonylurea should be stopped to prevent hypoglycaemia.

👍 👎 Discuss (9) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

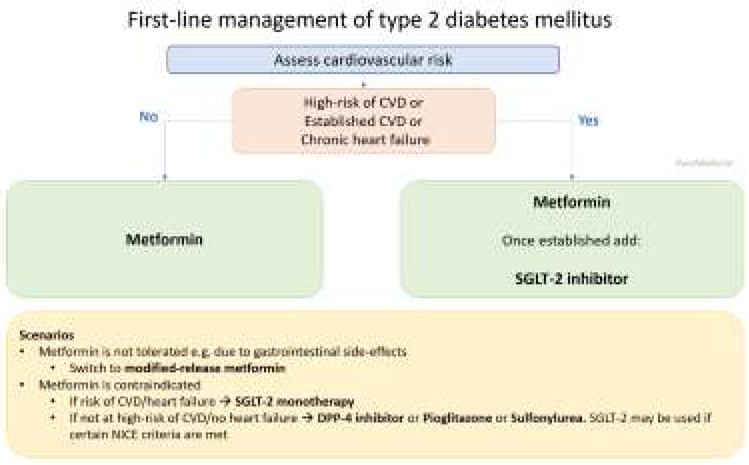
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

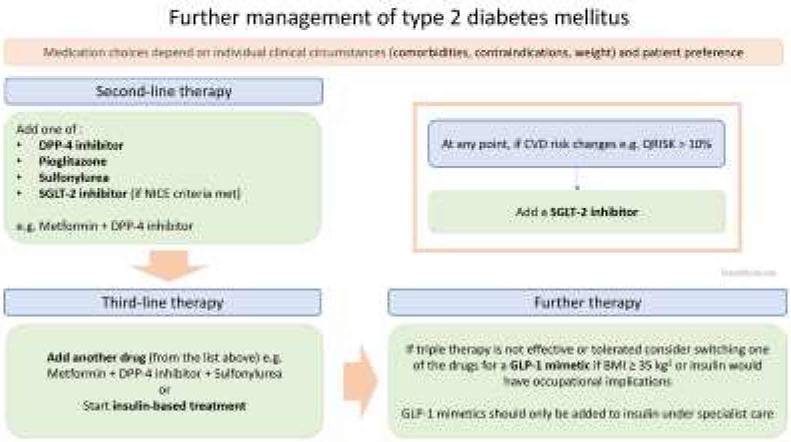
- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:

    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
• DPP-4 inhibitor
• Pioglitazone
• Sulfonylurea
• SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg/m^2 or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

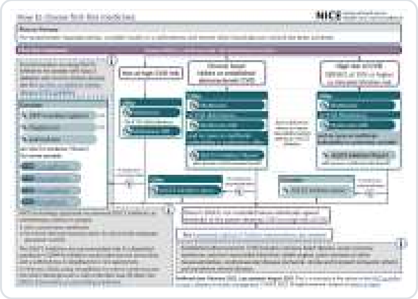
Starting insulin

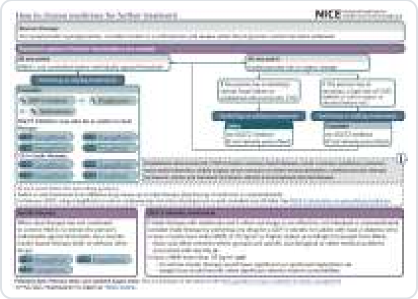
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

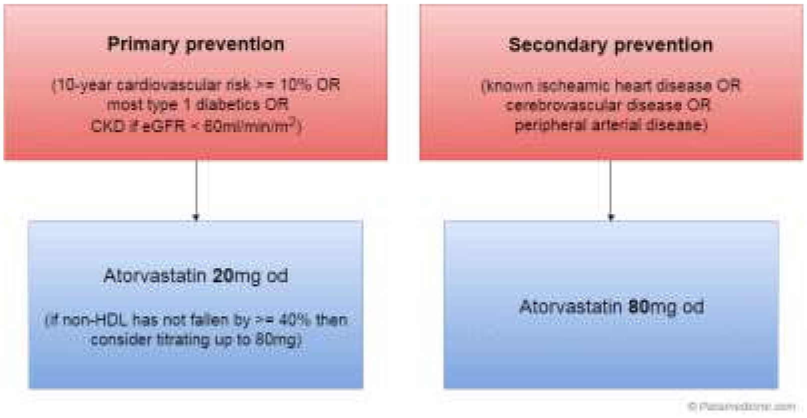
	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od

© Passmedicine.com

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

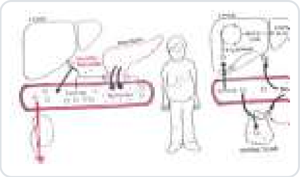
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

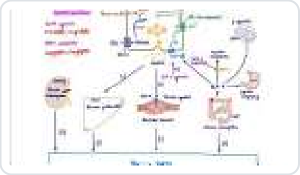
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **58.8%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓
7 ✗
8 ✓

9	✓
10	✗
11	✗
12	✗
13	✓
14	✓
15	✓
16	✗
17	✓

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Back to top

A 80-year-old patient was referred to Accident and Emergency after being found unresponsive in his home. He had just completed a course of antibiotics for a chest infection. He had not been seen for the preceding 36 hours. He had a past medical history of hypertension and type two diabetes.

His medication included Metformin, Gliclazide, Humulin M3 insulin twice a day, Ramipril and Bendroflumethiazide.

His initial examination revealed. Blood pressure 104/53, heart rate 103 beats per minute, respiratory rate 24 and oxygen saturations 90% on air. He had inspiratory crackles on his left lower lung zone. He had sunken eyes, capillary refill time of four seconds and no lower limb swelling. GCS 13 out of 15.

Initial blood tests;

Hb	11.0 g/dL
WCC	21.4 *10^9/l
Platelets	189 *10^9/l
CRP	340 mg/L
Na+	149 mmol/l
K+	4.4mmol/l
Ur	28 mmol/l
Cr	180 µmol/l
Glucose	54mmol/l

ABG on air

pH	7.32
pCO2	3.7kPa

pO2	9kPa
HCO3	18 mmol/l
Lactate	2.4mmol/l

Urine dipstick analysis - ++ glucose, - WCC, - leucocytes, + ketones

The patient was treated with oxygen, intravenous antibiotics for a chest infection and prophylactic low molecular weight heparin. They were treated with the local diabetic ketoacidosis protocol with IV inulin sliding scale and IV fluids 5500ml in 24 hours.

His repeat bloods 12 hours later were;

Na+	132 mmol/l
K+	3.9 mmol/l
Ur	12 mmol/l
Cr	110 μmmol/l
Glucose	5 mmol/l
HCO3	24 mmol/l
Lactate	1.7 mmol/l
CRP	270mg/l

The patient developed a grand mal seizure. His Glasgow coma scale remained 10 an hour after the seizure.

What is the most likely cause of his neurological deterioration?

- ○ Hypoglycaemia ×
- ○ Intracranial venous sinus thrombosis ×
- ○ Cerebral oedema ×
- ○ Sepsis ×

○ Renal failure ✕

Submit answer

Reference ranges

Score: **58.8%**

1	✓
2	✓
3	✕
4	✓
5	✕
6	✓
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13	✓
14	✓
15	✓
16	✕
17	✓
18	-

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ABG on air

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Ur	12 mmol/l
Cr	110 µmmol/l
Glucose	5 mmol/l
HCO3	24 mmol/l
Lactate	1.7 mmol/l
CRP	270mg/l

The patient developed a grand mal seizure. His Glasgow coma scale remained 10 an hour after the seizure.

What is the most likely cause of his neurological deterioration?

	Hypoglycaemia	4%
🟥	Intracranial venous sinus thrombosis	2%
🟩	Cerebral oedema	91%
	Sepsis	2%

Renal failure 0%

All the answers are potentially associated with hyperosmolar hyperglycaemic coma. The current guidance advises treatment initially with normal saline intravenous rehydration. The target reduction in osmolality is 3-8 mosmol/kg an hour. Only if this target is not being met and the glucose level not reducing sufficiently is insulin to be started.

This patient developed cerebral oedema secondary to rapid reduction in serum osmolality.

Discuss (7) Improve

Next question

Hyperosmolar hyperglycaemic state

Hyperosmolar hyperglycaemic state (HHS) is a medical emergency that can be difficult to manage and has a significant associated mortality (up to 20%). Hyperglycaemia results in osmotic diuresis, severe dehydration, and electrolyte deficiencies. HHS typically presents in the elderly with type 2 diabetes mellitus (T2DM).

Pathophysiology

- hyperglycaemia → ↑ serum osmolality → osmotic diuresis → severe volume depletion

Precipitating factors

- intercurrent illness
- dementia
- sedative drugs

Clinical features

- whilst DKA presents within hours of onset, HHS comes on over many days, and consequently, the dehydration and metabolic disturbances may be more extreme

- consequences of volume loss
  - clinical signs of dehydration
  - polyuria
  - polydipsia
- systemic
  - lethargy
  - nausea and vomiting
- neurological
  - altered level of consciousness
  - focal neurological deficits
- haematological
  - hyperviscosity (may result in myocardial infarctions, stroke and peripheral arterial thrombosis)

There are no precise **diagnostic criteria** but the following are typically seen

- hypovolaemia
- marked hyperglycaemia (>30 mmol/L)
- significantly raised serum osmolarity (> 320 mosmol/kg)
  - can be calculated by: 2 * Na^+ + glucose + urea
- no significant hyperketonaemia (<3 mmol/L)
- no significant acidosis (bicarbonate > 15 mmol/l or pH > 7.3 - acidosis can occur due to lactic acidosis or renal impairment)

Management

- fluid replacement
  - fluid losses in HHS are estimated to be between 100 - 220 ml/kg
  - IV 0.9% sodium chloride solution
  - typically given at 0.5 - 1 L/hour depending on clinical assessment
  - potassium levels should be monitored and added to fluids depending on the level
- insulin
  - should not be given unless blood glucose stops falling while giving IV fluids
- venous thromboembolism prophylaxis
  - patients are at risk of thrombosis due to hyperviscosity

Complications

- vascular complications may occur due to hyperviscosity:
    - such as myocardial infarction
    - stroke

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Diabetes Societies 👍 20 👎 13

The management of the hyperosmolar hyperglycaemic state (HHS)

Suggest link Report broken link

Score: **55.6%**

1 ✓

2 ✓

3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
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13	✓
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17	✓
18	✗

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Question 19 of 101

An 82-year-old female presents to clinic with her daughter complaining of a four month of history of urinary incontinence. She explains that she has not previously had problems with continence. Her only past medical history include hypertension and angina. Now, she is incontinent of urine only when she laughs or coughs. At times, she reports sudden urges to urinate at all times during the day, resulting in a leak when she is unable to reach the toilet in time. This is significantly impacted on her sleep as well as it is increasingly frequent at night. The patient has reduced her caffeine intake already and has commenced 'bladder training' recommended by her GP. What additional management would you commence?

- ○ Pelvic floor exercises only ×
- ○ Pelvic floor exercises and tolterodine ×
- ○ Pelvic floor exercises and duloxetine ×
- ○ Pelvic floor exercises and desmopressin ×
- ○ Long term urinary catheter ×

Submit answer

Reference ranges

Score: **55.6%**

1 ✓

2	✓
3	✗
4	✓
5	✗
6	✓
7	✗
8	✓
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17	✓
18	✗
19	-

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Question 19 of 101 ✓

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	Pelvic floor exercises only	20%
✓	Pelvic floor exercises and tolterodine	32%
	Pelvic floor exercises and duloxetine	42%
	Pelvic floor exercises and desmopressin	5%
	Long term urinary catheter	1%

The question concentrates on the management of an elderly patient with mixed stress incontinence and overactive bladder syndrome. In the context of mixed urinary incontinence, pharmacological therapies should be offered in addition to conservative therapies such as pelvic floor exercises. A long-term catheter in whom persistent incontinence causes skin wounds, the patient requires such significant care that continuous changing of bed linen and clothes would be required, chronic retention with a risk of renal impairment (and inability to self-catheterise) or if the patient opts for the catheterisation. The 2013 NICE guidelines recommends first line pharmacological treatment for mixed incontinence or overactive bladder syndrome alone is either tolterodine, oxybutynin or darifenacin1. Duloxetine should only be used if the patient does not tolerate first line therapy and is not a candidate for surgery. Desmopressin can reduce nocturia. However, its effects in platelet activation contradicts its use in ischaemic heart disease.

1. NICE Guideline 171. Urinary incontinence. The management of urinary incontinence in women. September 2013

👍 👎 Discuss (8) Improve

Next question

Urinary incontinence ★

Urinary incontinence (UI) is a common problem, affecting around 4-5% of the population. It is more common in elderly females.

Risk factors

- advancing age
- previous pregnancy and childbirth
- high body mass index
- hysterectomy
- family history

Classification

- overactive bladder (OAB)/urge incontinence
  - due to detrusor overactivity
  - the urge to urinate is quickly followed by uncontrollable leakage ranging from a few drops to complete bladder emptying
- stress incontinence: leaking small amounts when coughing or laughing
- mixed incontinence: both urge and stress
- overflow incontinence: due to bladder outlet obstruction, e.g. due to prostate enlargement
- functional incontinence
  - comorbid physical conditions impair the patient's ability to get to a bathroom in time

    - causes include dementia, sedating medication and injury/illness resulting in decreased ambulation

Initial investigation

- bladder diaries should be completed for a minimum of 3 days
- vaginal examination to exclude pelvic organ prolapse and ability to initiate voluntary contraction of pelvic floor muscles ('Kegel' exercises)
- urine dipstick and culture
- urodynamic studies
    - not routinely required before starting initial conservative treatment for urinary incontinence
    - may be considered if the diagnosis is uncertain, mixed incontinence is suspected or surgical intervention is planned

Management depends on whether urge or stress UI is the predominant picture

If urge incontinence is predominant:

- bladder retraining (lasts for a minimum of 6 weeks, the idea is to gradually increase the intervals between voiding)
- bladder stabilising drugs: antimuscarinics are first-line
    - NICE recommend oxybutynin (immediate release), tolterodine (immediate release) or darifenacin (once daily preparation)
    - avoid immediate-release oxybutynin in frail older women
    - solifenacin is an alternative antimuscarinic agent
- mirabegron (a beta-3 agonist) may be useful if there is concern about anticholinergic side-effects in frail elderly patients
- if troublesome nocturia is a feature, desmopressin may be considered
- for women with overactive bladder that has not responded to non-surgical management:
    - if urodynamic investigations determine that detrusor overactivity is causing her overactive bladder symptoms then consider botulinum toxin type A bladder wall injection

If stress incontinence is predominant:

- lifestyle measures, e.g. weight reduction, reducing caffeine intake
- pelvic floor muscle training
    - NICE recommend at least 8 contractions performed 3 times per day for a minimum of 3 months
- surgical procedures include:
    - mid-urethral tape procedures (still recommended by NICE but only after discussing risks and benefits)
    - intramural bulking agent injections
    - colposuspension
- duloxetine may be offered to women if they decline surgical procedures
    - a combined noradrenaline and serotonin reuptake inhibitor
    - mechanism of action: increased synaptic concentration of noradrenaline and serotonin within the pudendal nerve → increased stimulation of urethral striated muscles within the sphincter → enhanced contraction

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 23 👎 30

2019 urinary incontinence guidelines

Suggest link Report broken link

Score: **57.9%**

1	✓
2	✓
3	✗
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17	✓
18	✗
19	✓

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You are called to review a 43-year-old woman who has received 48 hours of treatment for diabetic ketoacidosis. She has had diarrhoea and abdominal discomfort for the past 12 hours is now become increasingly confused with visual hallucinations and aggressive behaviour.

She has a history of type 1 diabetes mellitus and Grave's disease which has been in remission and not requiring treatment.

Observations: SpO2 97% room air, respiratory rate 18 breaths per minute, heart rate 145 beats per minute, blood pressure 170/110 mmHg, temperature 40.2°C.

On examination, she has generalised abdominal pain, mild pitting oedema, and an irregularly irregular pulse.

Hb	135 g/L	Male: (135-180) Female: (115 - 160)
Platelets	422 * 10^9/L	(150 - 400)
WBC	8.5 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.0 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	65 µmol/L	(55 - 120)
CRP	24 mg/L	(< 5)
TSH	0.4 mIU/L	(0.5-5)
FT4	32 pmol/L(12-22)	

What is the most appropriate acute treatment for the likely underlying cause of this patient's current presentation?

○ 300mg aspirin ✕

- ○ Carbimazole ×
- ○ IV antibiotics ×
- ○ Propylthiouracil ×
- ○ Thyroidectomy ×

Submit answer

Reference ranges

Score: **57.9%**

1	✓
2	✓
3	×
4	✓
5	×
6	✓
7	×
8	✓
9	✓
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18	×

19	✓
20	-

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You are called to review a 43-year-old woman who has received 48 hours of treatment for diabetic ketoacidosis. She has had diarrhoea and abdominal discomfort for the past 12 hours is now become increasingly confused with visual hallucinations and aggressive behaviour.

She has a history of type 1 diabetes mellitus and Grave's disease which has been in remission and not requiring treatment.

Observations: SpO2 97% room air, respiratory rate 18 breaths per minute, heart rate 145 beats per minute, blood pressure 170/110 mmHg, temperature 40.2°C.

On examination, she has generalised abdominal pain, mild pitting oedema, and an irregularly irregular pulse.

Hb	135 g/L	Male: (135-180) Female: (115 - 160)
Platelets	422 * 10^9/L	(150 - 400)
WBC	8.5 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.0 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	65 µmol/L	(55 - 120)
CRP	24 mg/L	(< 5)
TSH	0.4 mIU/L	(0.5-5)
FT4	32 pmol/L(12-22)	

What is the most appropriate acute treatment for the likely underlying cause of this patient's current presentation?

300mg aspirin 3%

🟥	Carbimazole	24%
	IV antibiotics	4%
🟩	Propylthiouracil	68%
	Thyroidectomy	1%

In thyroid storm, treat acutely with propylthiouracil rather than carbimazole or surgery

Important for me Less important

The stem describes a classic case of thyroid storm. It is most commonly seen in patients with a background of Grave's disease but can occur in other scenarios too. There is usually a precipitating insult such as diabetic ketoacidosis or a heart attack.

The likelihood of a thyroid storm can be calculated using the Burch and Wartofksy thyroid storm diagnostic criteria. This attributes points to the presence of pyrexia, tachycardia, signs of heart failure, confusion, coma, seizures, gastrointestinal disturbance etc.

In this case, the patient scores 90. A score of >45 indicates a likely thyroid storm. The patient also has a suppressed TSH level with raised T4, this cements the diagnosis.

In an ideal situation treatment for presumed thyroid storm should not be delayed whilst awaiting blood results as this condition has significant mortality (>10%). Blood tests may be in keeping with hyperthyroidism, but there is no specific cut-off to diagnose thyroid storm, the diagnosis is made on clinical findings.

Propylthiouracil is the correct answer. Patients should be assessed with an ABCDE approach and stabilised. IV fluids and cooling are important aspects of treatment. Tachyarrhythmias should also be managed according to local guidelines. The most appropriate treatment for the underlying thyroid storm is propylthiouracil. This inhibits thyroid hormone synthesis and also inhibits the conversion of T4 to T3.

Carbimazole is incorrect. Although carbimazole can be used, propylthiouracil is the traditionally preferred treatment as it has a faster onset of action and also inhibits the conversion of fT4 to T3.

Thyroidectomy is incorrect. This can be used as a definitive treatment for Grave's disease and can also be used in the acute setting if a patient has had a previous poor reaction to thionamides such as propylthiouracil. Usually, the first-line treatment would be stabilisation with beta-blockers, supportive treatment and oral thionamides.

IV antibiotics is incorrect. In this case, the patient is pyrexial and has new-onset atrial fibrillation with a rapid ventricular response. Diarrhoea and abdominal pain could indicate an intra-abdominal infection. It would not be unusual for the patient to receive IV antibiotics in this scenario to cover for potential infection, but given the blood results here - normal white cell count, normal CRP, and deranged thyroid function - we can be quite sure of a diagnosis of thyroid storm which wouldn't respond to IV antibiotics. The concomitant atrial fibrillation with signs of heart failure (pedal oedema), extreme pyrexia, and delirium, are better explained by thyroid storm.

300mg aspirin is incorrect. This would be the treatment of stroke. There is an altered conscious level with behavioural changes and pyrexia, both of which can be seen in acute stroke. There are, however, no focal neurological findings described and a stroke would not explain the change in bowel habit and abdominal pain.

👍 👎 Discuss (2) Improve

Next question

Thyroid storm ★

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:

- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:

- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:

- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

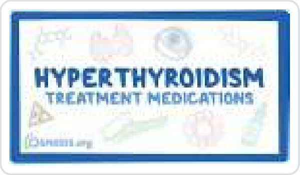
Extended textbook

Media

THYROID STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1


HYPERTHYROIDISM
TREATMENT MEDICATIONS

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **55%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓
7 ✗
8 ✓

9	✓
10	✗
11	✗
12	✗
13	✓
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20	✗

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Question 21 of 101

A 28-year-old woman presents with daily intractable vomiting. She is 10 weeks pregnant with 3+ ketones in her urine. Initially, she tried cyclizine from her general practitioner with little success. You suspect hyperemesis gravidarum. Apart from rehydration and anti-emetics, what also should be prescribed?

- ○ Thiamine ×
- ○ Carbohydrate replacement with 5% dextrose ×
- ○ Niacin ×
- ○ Vitamin B12 ×
- ○ Folate ×

Submit answer

Reference ranges

Score: **55%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓

Dr. Jamil Alrajhi

7	✗
8	✓
9	✓
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18	✗
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20	✗
21	-

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← Question 21 of 101 ✗ ⚐ →

A 28-year-old woman presents with daily intractable vomiting. She is 10 weeks pregnant with 3+ ketones in her urine. Initially, she tried cyclizine from her general practitioner with little success. You suspect hyperemesis gravidarum. Apart from rehydration and anti-emetics, what also should be prescribed?

🟩	Thiamine	57%
	Carbohydrate replacement with 5% dextrose	12%
	Niacin	8%
	Vitamin B12	4%
🟥	Folate	18%

Nausea and vomiting are both common in pregnancy, affecting 50-80% of pregnant women. Hyperemesis gravidarum occurs in 0.3-3% of pregnancies. It is a diagnosis of exclusion and other causes of vomiting should be considered: urinary tract infection, endocrine causes (thyrotoxicosis, diabetic ketoacidosis etc), surgical causes and drugs such as iron supplements.

The Royal College of Obstetrics and Gynaecology (RCOG) guidelines state:
Thiamine supplementation (either oral or intravenous) should be given to all women admitted with prolonged vomiting, especially before administration of dextrose or parenteral nutrition.
(https:www.rcog.org.uk/globalassets/documents/guidelines/green-top-guidelines/gtg69-hyperemesis.pdf)

A 5% dextrose intravenous infusion may precipitate Wernicke's encephalopathy. In addition, patients are frequently hyponatraemic and therefore sodium chloride 0.9% is most appropriate (+/- potassium replacement).

Folic acid (400 micrograms) daily is recommended for all women prior to conception and up to the 13th week of pregnancy. Women who are deemed higher risk of spina bifida are advised to take 5mg daily. This is to prevent neural tube defects but has no role in hyperemesis gravidarum.

Vitamin B12 deficiency is not of clinical concern in this scenario.

Niacin deficiency leads to pellagra. Hyperemesis gravidarum is a self-limiting condition associated with pregnancy and therefore niacin deficiency is not of clinical concern here.

👍 👎 💬 Discuss (7) Improve

Next question

Hyperemesis gravidarum ★

Whilst the majority of women experience nausea (previously termed 'morning sickness') during the early stages of pregnancy it can become problematic in a minority of cases. The Royal College of Obstetricians and Gynaecologists (RCOG) now use the term 'nausea and vomiting of pregnancy' (NVP) to describe troublesome symptoms, with hyperemesis gravidarum being the extreme form of this condition.

It occurs in around 1% of pregnancies and is thought to be related to raised beta hCG levels. Hyperemesis gravidarum is most common between 8 and 12 weeks but may persist up to 20 weeks.

Risk factors
- increased levels of beta-hCG
    - multiple pregnancies
    - trophoblastic disease
- nulliparity
- obesity

- family or personal history of NVP

Smoking is associated with a decreased incidence of hyperemesis.

Referral criteria for nausea and vomiting in pregnancy

NICE recommend considering admission in the following situations:
- Continued nausea and vomiting and is unable to keep down liquids or oral antiemetics
- Continued nausea and vomiting with ketonuria and/or weight loss (greater than 5% of body weight), despite treatment with oral antiemetics
- A confirmed or suspected comorbidity (for example she is unable to tolerate oral antibiotics for a urinary tract infection)

They also recommend having a lower threshold for admission to hospital if the woman has a co-existing condition (for example diabetes) that may be adversely affected by nausea and vomiting.

Hyperemesis gravidarum

The Royal College of Obstetricians and Gynaecologists (RCOG) recommend that the following triad is present before diagnosis of hyperemesis gravidarum: RCOG
- 5% pre-pregnancy weight loss
- dehydration
- electrolyte imbalance

Validated scoring systems such as the Pregnancy-Unique Quantification of Emesis (PUQE) score can be used to classify the severity of NVP.

Management
- simple measures
    - rest and avoid triggers e.g. odours
    - bland, plain food, particularly in the morning

    - ginger
    - P6 (wrist) acupressure
- first-line medications
    - antihistamines: oral cyclizine or promethazine
    - phenothiazines: oral prochlorperazine or chlorpromazine
    - combination drug doxylamine/pyridoxine: pyridoxine (vitamin B6) monotherapy is actually used commonly outside of the UK as a first-line treatment for NVP. However, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines
- second-line medications
    - oral ondansetron: ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate. The Medicines and Healthcare products Regulatory Agency (MHRA) advise that if ondansetron is used then these risks should be discussed with the pregnant woman
    - oral metoclopramide or domperidone: metoclopramide may cause extrapyramidal side effects. It should therefore not be used for more than 5 days
- admission may be needed for IV hydration
    - see criteria above for referral/admission
    - normal saline with added potassium is used to rehydrate

Women with hyperemesis gravidarum may develop dehydration, weight loss and electrolyte imbalances. Other complications include:

- acute kidney injury
- Wernicke's encephalopathy
- oesophagitis, Mallory-Weiss tear
- venous thromboembolism
- fetal outcome
    - studies generally show little evidence of adverse outcomes for birth weight/other markers for mild-moderate symptoms
    - severe NVP resulting in multiple admissions and failure to 'catch-up' weight gain may be linked to a small increase in preterm birth and low birth weight

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Obstetricians and Gynaecologists 👍 11 👎 13

2016 The Management of Nausea and Vomiting of Pregnancy and Hyperemesis Gravidarum

Clinical Knowledge Summaries 👍 8 👎 4

CKS - NICE vomiting in pregnancy guidance summary

Suggest link

Report broken link

Score: **52.4%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
6 ✓

7	✗
8	✓
9	✓
10	✗
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17	✓
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21	✗

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Question 22 of 101

A 45-year-old woman presents to the emergency department feeling very hot, weight loss and nausea. She has a past medical history of cardiomyopathy for which she an ICD inserted and takes amiodarone due to recurrent episodes of ventricular tachycardia. Blood tests show an undetectable thyroid stimulating hormone. She also has rheumatoid arthritis. She has previously had an angiogram showing no evidence of coronary artery disease. She takes methotrexate, aspirin, paracetamol, omeprazole, warfarin and bisoprolol. She is suspected of having acute thyrotoxicosis. What is the most appropriate action in regards to her current medication in addition to stopping amiodarone?

○ Stop aspirin ✕

○ Stop omeprazole ✕

○ Stop bisoprolol ✕

○ Stop warfarin ✕

○ Stop paracetamol ✕

Submit answer

Reference ranges

Score: **52.4%**

1 ✓

2 ✓

3	✗
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22	-

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Back to top

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🟩	Stop aspirin	30%
🟥	Stop omeprazole	29%
	Stop bisoprolol	14%
	Stop warfarin	26%
	Stop paracetamol	2%

In acute thyrotoxicosis, stop aspirin as it can worsen the storm by displacing T4 from thyroid binding globulin

Important for me Less important

This patient with acute thyrotoxicosis should have amiodarone, the likely cause, of her hyperthyroidism stopped. In addition, aspirin should be stopped. Aspirin binds to thyroxine-binding globulin and displaces bound T4, thereby increasing the levels of free T4.

Discuss (7) Improve

Next question

Thyrotoxicosis: features

General

- Weight loss
- 'Manic', restlessness
- Heat intolerance

Cardiac

- palpitations, tachycardia
- high-output cardiac failure may occur in elderly patients, a reversible cardiomyopathy can rarely develop

Skin

- Increased sweating
- Pretibial myxoedema: erythematous, oedematous lesions above the lateral malleoli
- Thyroid acropachy: clubbing

Gastrointestinal

- Diarrhoea

Gynaecological

- Oligomenorrhea

Neurological

- Anxiety
- Tremor

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

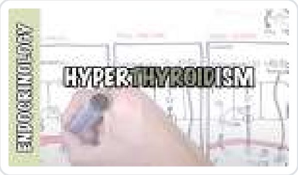
Royal College of Physicians 👍 0 👎 1

2017 Thyrotoxicosis â€“ investigation and management

Suggest link

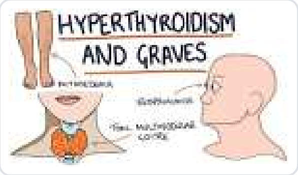
Report broken link

Media


ENDOCRINOLOGY
HYPERTHYROIDISM

Hyperthyroidism Overview (causes, pathophysiology)

Armando Hasudungan - YouTube 👍 2 👎 0


HYPERTHYROIDISM
AND GRAVES

Understanding Hyperthyroidism and Graves' Disease

Zero to Finals - YouTube 👍 1 👎 0

Report broken media

Score: **50%**

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A 62-year-old taxi driver is reviewed in the diabetes clinic some 6 weeks after suffering from an inferior myocardial infarction, for which he was stented. Current medication for control of blood sugar is metformin 1g twice a day. Other medication of note includes ramipril and indapamide. His blood pressure is 139/85 mmHg, his pulse is 84 beats per minute and regular. There are bilateral basal crackles on auscultation of the chest consistent with cardiac failure.

Na^+	140 mmol/l
K^+	4.5 mmol/l
Urea	7.2 mmol/l
Creatinine	112 µmol/l
HbA1c	64 mmol/mol

Which of the following is the most appropriate intervention with respect to his glycaemic control?

- ○ Add empagliflozin ×
- ○ Add gliclazide ×
- ○ Add insulin glargine ×
- ○ Add pioglitazone ×
- ○ Add saxagliptin ×

Submit answer

Reference ranges

Score: **50%**

1	✓
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7	✗
8	✓
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19	✓
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Question 23 of 101 ✓

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Na^+	140 mmol/l
K^+	4.5 mmol/l
Urea	7.2 mmol/l
Creatinine	112 µmol/l
HbA1c	64 mmol/mol

Which of the following is the most appropriate intervention with respect to his glycaemic control?

[✓ Add empagliflozin]	95%
Add gliclazide	2%
Add insulin glargine	1%
Add pioglitazone	1%
Add saxagliptin	2%

SGLT-2 inhibition is the best option for glucose control in a patient with IHD and heart failure failing glycaemic control on metformin because there is good evidence for outcome benefit across the class

Important for me Less important

This patient has a history of diabetes and cardiac failure after a myocardial infarction. Evidence in this patient group is strong for a putative benefit of SGLT-2 inhibitors. In particular both empagliflozin and canagliflozin have reported outcomes data which demonstrates a reduction in mortality of around 1/3rd. Their outcome trials also demonstrate a reduction in episodes of symptomatic cardiac failure and reduced progression of microvascular complications of diabetes.

The other options are all inappropriate. Adding gliclazide is associated with increased risk of hypoglycaemia, weight gain, and is not associated with positive cardiovascular outcomes. In particular, given his occupation as a taxi driver, hypoglycaemia should be avoided. Insulin glargine may promote significant weight gain, can worsen cardiac failure and does also cause hypoglycaemia. Pioglitazone promotes weight gain and fluid retention and is therefore contraindicated in cardiac failure. Saxagliptin was associated with an increase in cardiac failure in the SAVOR-TIMI cardiac outcome trial.

http://www.nejm.org/doi/full/10.1056/NEJMoa1504720#t=article

👍 👎 Discuss (5) Improve

Next question

SGLT-2 inhibitors ★

SGLT-2 inhibitors reversibly inhibit sodium-glucose co-transporter 2 (SGLT-2) in the renal proximal convoluted tubule to reduce glucose reabsorption and increase urinary glucose excretion.

Examples include canagliflozin, dapagliflozin and empagliflozin.

Important adverse effects include
- urinary and genital infection (secondary to glycosuria). Fournier's gangrene has also been reported
- normoglycaemic ketoacidosis

- increased risk of lower-limb amputation: feet should be closely monitored

Patients taking SGLT-2 drugs often lose weight, which can be beneficial in type 2 diabetes mellitus.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

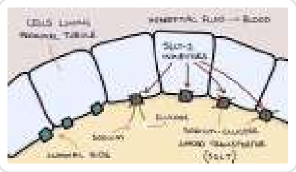
Links

BNF Dapagliflozin	👍 14 👎 15

Suggest link

Report broken link

Media


CELLS LINING PROXIMAL TUBULE
INTERSTITIAL FLUID → BLOOD
SGLT-2 INHIBITORS
GLUCOSE
SODIUM
LUMINAL SIDE
SODIUM-GLUCOSE LINKED TRANSPORTER (SGLT)

How does Dapagliflozin work? Understanding SGLT2 inhibitors.

Zero To Finals - YouTube 👍 6 👎 0

Report broken media

Score: **52.2%**

1	✓
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23	✓

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A 40-year-old school teacher presented to the medical assessment unit with a 2-month history of polyuria and elevated blood glucose levels. There was no family history of diabetes and she was not currently on any medication. On examination, her body mass index was 22 kg/m^2 with normal general physical and systemic examination. Urine was negative for ketones.

Blood glucose	16.5 mmol/l
pH	7.40
HCO_3	25 mmol/l
Na^+	140 mmol/l
K^+	3.7 mmol/l

Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

● Oral glucose tolerance test ×

○ Serum ferritin and total iron binding capacity ×

○ Mitochondrial gene mutation (A3243G) ×

○ Anti-GAD antibody ×

○ Toxicology screen ×

Submit answer

Reference ranges

Score: **52.2%**

1	✓
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18	✗
19	✓
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22	✗
23	✓
24	-

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pH	7.40
HCO_3	25 mmol/l
Na^+	140 mmol/l
K^+	3.7 mmol/l

Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

🟥	Oral glucose tolerance test	27%
	Serum ferritin and total iron binding capacity	5%
	Mitochondrial gene mutation (A3243G)	10%
🟩	Anti-GAD antibody	56%
	Toxicology screen	1%

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of T2DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies)

Important for me Less important

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of type 2 DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies). Most of these patients are in the age group of 30-50 years, may or may not have a normal BMI and may present with elevated blood glucose levels. These patients can be managed initially with oral hypoglycaemic agents, although the β-cell function may decline over months to a few years, necessitating a relatively early requirement for insulin use.

👍 👎 Discuss (7) Improve

Next question

Diabetes mellitus (type 2): diagnosis ★

The diagnosis of type 2 diabetes mellitus can be made by either a plasma glucose or a HbA1c sample. Diagnostic criteria vary according to whether the patient is symptomatic (polyuria, polydipsia etc) or not.

If the patient is symptomatic:

- fasting glucose greater than or equal to 7.0 mmol/l
- random glucose greater than or equal to 11.1 mmol/l (or after 75g oral glucose tolerance test)

If the patient is asymptomatic the above criteria apply but must be demonstrated on two separate occasions.

When HbA1c is used for the diagnosis of diabetes:

- a HbA1c of greater than or equal to 48 mmol/mol (6.5%) is diagnostic of diabetes mellitus
- a HbAlc value of less than 48 mmol/mol (6.5%) does not exclude diabetes (i.e. it is not as sensitive as fasting samples for detecting diabetes)
- in patients without symptoms, the test must be repeated to confirm the diagnosis

- it should be remembered that misleading HbA1c results can be caused by increased red cell turnover (see below)

Conditions where HbA1c may not be used for diagnosis:

- haemoglobinopathies
- haemolytic anaemia
- untreated iron deficiency anaemia
- suspected gestational diabetes
- children
- HIV
- chronic kidney disease
- people taking medication that may cause hyperglycaemia (for example corticosteroids)

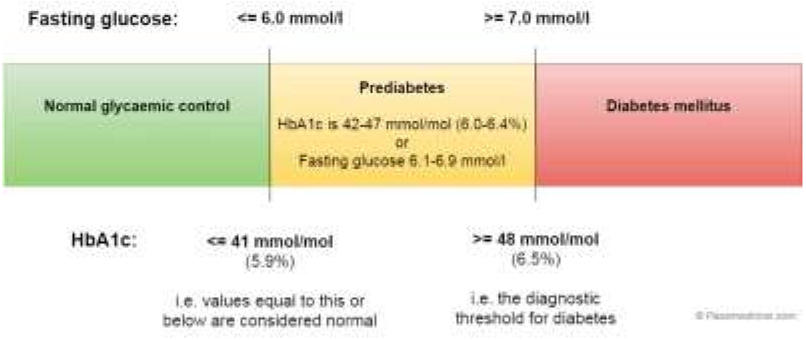

Fasting glucose:
<= 6.0 mmol/l
>= 7.0 mmol/l
Normal glycaemic control
Prediabetes
HbA1c is 42-47 mmol/mol (6.0-6.4%)
or
Fasting glucose 6.1-6.9 mmol/l
Diabetes mellitus
HbA1c:
<= 41 mmol/mol
(5.9%)
i.e. values equal to this or below are considered normal
>= 48 mmol/mol
(6.5%)
i.e. the diagnostic threshold for diabetes

Diagram showing the spectrum of diabetes diagnosis

Impaired fasting glucose and impaired glucose tolerance

A fasting glucose greater than or equal to 6.1 but less than 7.0 mmol/l implies impaired fasting glucose (IFG)

Impaired glucose tolerance (IGT) is defined as fasting plasma glucose less than 7.0 mmol/l and OGTT 2-hour value greater than or equal to 7.8 mmol/l but less than 11.1 mmol/l

Diabetes UK suggests:

- 'People with IFG should then be offered an oral glucose tolerance test to rule out a diagnosis of diabetes. A result below 11.1 mmol/l but above 7.8 mmol/l indicates that the person doesn't have diabetes but does have IGT.'

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 18 👎 20

Type 2 diabetes mellitus: diagnosis

Diabetes UK 👍 23 👎 12

New diagnostic criteria for diabetes

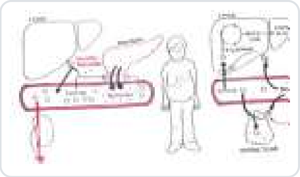
NICE 👍 21 👎 10

2022 Type 2 diabetes guidelines

Suggest link

Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **50%**

1	✓
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4	✓
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22 ✗

23 ✓

24 ✗

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Question 25 of 101

A 32-year-old man is referred to the endocrinology clinic with a 6-month history of excessive thirst and passing large volumes of urine, including waking several times at night to void. He has no significant past medical history and takes no regular medications. His serum sodium is mildly elevated on recent blood tests. A water deprivation test is organised. The following laboratory values are obtained:

Plasma osmolality (final)	305 mOsm/kg	(275 - 295)
Urine osmolality (final)	180 mOsm/kg	(500 - 800)
Urine osmolality after desmopressin	185 mOsm/kg	(500 - 800)

Which of the following is the most likely diagnosis?

- ○ Nephrogenic diabetes insipidus ×
- ○ Cranial diabetes insipidus ×
- ○ Primary polydipsia ×
- ○ Partial cranial diabetes insipidus ×
- ○ Syndrome of inappropriate antidiuretic hormone secretion (SIADH) ×

Submit answer

Reference ranges

Score: **50%**

1	✓
2	✓
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23	✓
24	✗
25	-

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Urine osmolality (final)	180 mOsm/kg	(500 - 800)
Urine osmolality after desmopressin	185 mOsm/kg	(500 - 800)

Which of the following is the most likely diagnosis?

Nephrogenic diabetes insipidus	89%
Cranial diabetes insipidus	5%
Primary polydipsia	4%
Partial cranial diabetes insipidus	0%
Syndrome of inappropriate antidiuretic hormone secretion (SIADH)	1%

Water deprivation test: nephrogenic DI

- urine osmolality after fluid deprivation: low
- urine osmolality after desmopressin: low

Important for me Less important

Nephrogenic diabetes insipidus is the correct answer. This condition arises when the kidneys are unresponsive to antidiuretic hormone (ADH). In this scenario, despite water deprivation leading to increased plasma osmolality, the urine remains inappropriately dilute both before and after administration of desmopressin. The failure to concentrate urine after desmopressin administration distinguishes nephrogenic DI from cranial DI, where a response would be expected.

Cranial diabetes insipidus occurs due to deficient production or release of ADH from the posterior pituitary. In cranial DI, urine osmolality remains low during water deprivation but increases significantly after desmopressin administration because exogenous ADH can act on functioning renal receptors. This patient's lack of response to desmopressin makes this diagnosis less likely.

Primary polydipsia, also known as psychogenic polydipsia, involves excessive water intake suppressing endogenous ADH release and causing dilute urine initially; however, with water deprivation, these patients can usually concentrate their urine appropriately as endogenous ADH secretion resumes. In this case, there was no appropriate rise in urine osmolality with dehydration or desmopressin, arguing against primary polydipsia.

Partial cranial diabetes insipidus may present with some increase in urine concentration after desmopressin but not as marked as complete cranial DI. However, there should still be at least a partial response post-desmopressin; here, there is essentially none (<300 mOsm/kg), making partial cranial DI less plausible.

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) would typically present with hyponatraemia and concentrated urine despite hypo-osmolal plasma due to inappropriate ADH secretion; this patient has hypernatraemia and persistently dilute urine even after dehydration and desmopressin—findings inconsistent with SIADH.

👍 👎 💬 Discuss Improve

Next question

Water deprivation test ★

The water deprivation test is designed to help evaluate patients who have polydipsia.

Method
- prevent patient drinking water
- ask the patient to empty their bladder
- hourly urine and plasma osmolalities

	Starting plasma osm.	**Final urine osm.**	**Urine osm. post-DDAVP**
Normal	Normal	> 600	> 600
Psychogenic polydipsia	Low	> 400	> 400
Cranial DI	High	< 300	> 600
Nephrogenic DI	High	< 300	< 300

+ Q 123

Next question

B I A

Textbooks

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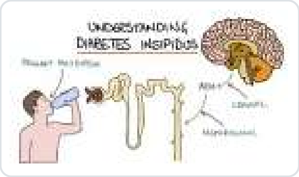
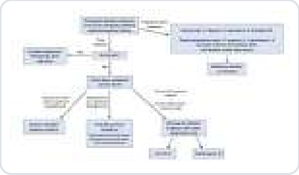
Extended textbook

Media


UNDERSTANDING DIABETES INSIPIDUS

[Understanding Diabetes Insipidus](#)

Zero to Finals - YouTube 👍 3 👎 0



[An Approach to Polyuria](#)

Strong Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **52%**

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25	✓

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Question 26 of 101 ✓

A 22-year-old student comes to the Emergency department with a cough productive of rusty coloured sputum. She has been suffering from increased shortness of breath, night sweats and fevers for the past 48 hours. Current medication includes daily hydrocortisone for congenital adrenal hyperplasia and the combined oral contraceptive pill. Current bloods are shown below:

Hb	131 g/l	Na^+	134 mmol/l
Platelets	201 * 10^9/l	K^+	4.1 mmol/l
WBC	14.9 * 10^9/l	Urea	7.0 mmol/l
Neuts	10.1 * 10^9/l	Creatinine	82 µmol/l
Lymphs	1.2 * 10^9/l	CRP	185 mg/l
Eosin	0.4 * 10^9/l		

Which of the following is the most appropriate way to manage her steroid hormone replacement?
How should you manage her steroid replacement?

Convert to 200mg hydrocortisone IV BD	4%
Increase the daily dose by 50%	29%
[✓ Increase the daily dose by 100%]	64%
Reduce the daily dose by 50%	2%
Keep the daily dose the same	1%

Patients with congenital adrenal hyperplasia who are managed with steroid hormone replacement should be managed in the same way as patients with Addison's disease. In other words, during a period of significant acute infection like the pneumonia here, the dose of corticosteroid should be doubled.

With the patient able to swallow, having presented early with her pneumonia, switching to IV hydrocortisone would represent excess steroid replacement and is not appropriate here. The other options including only small increases in steroid dose, reductions or maintaining the status quo, run the risk of adrenal crisis.

👍 | 👎 | 💬 Discuss (4) | Improve

Next question

Congenital adrenal hyperplasia ★

Congenital adrenal hyperplasia (CAH) refers to a group of autosomal recessive disorders that impair adrenal steroid biosynthesis. The deficiency in cortisol production leads to compensatory overproduction of adrenocorticotropic hormone (ACTH) by the anterior pituitary. Elevated ACTH levels increase the production of adrenal androgens, which can result in the virilization of female infants and affect genital development.

Cause
- 21-hydroxylase deficiency (90%)
  - impairs the conversion of 17-hydroxyprogesterone to 11-deoxycortisol, leading to cortisol deficiency and excess androgen production
- 11-beta hydroxylase deficihttps://www.passmedicine.com/database/dbnotes.php#ency (5%)
  - results in hypertension due to excess deoxycorticosterone
- 17-hydroxylase deficiency (very rare)
  - leads to mineralocorticoid excess with low androgen and estrogen levels

Clinical features

- the symptoms and signs of CAH vary depending on the specific enzyme deficiency and the severity of the disorder. Common clinical presentations include:
- virilization:
  - female infants may present with ambiguous genitalia due to excessive androgen exposure in utero.
  - male infants appear normal at birth, which can delay diagnosis.
- salt-wasting crisis:
  - this severe form occurs in about 75% of cases with 21-hydroxylase deficiency
  - it is characterized by dehydration, hypotension, and electrolyte imbalances, and can be life-threatening if not treated promptly.
- precocious puberty: Excess androgens can lead to early development of secondary sexual characteristics in both males and females.
- infertility: Adults with untreated CAH may experience fertility issues due to hormonal imbalances.
- height and growth abnormalities: Children with CAH often experience accelerated growth rates initially but may have a shorter adult stature due to early epiphyseal closure.

Diagnosis

- many countries screen newborns for CAH, looking for elevated levels of serum concentration of 17-hydroxyprogesterone (17OHP). This is however not yet done in the UK
- ACTH stimulation testing is used to confirm the diagnosis
  - evaluates the adrenal gland's response to ACTH, with abnormal increases in 17OHP indicating CAH

Management

- primarily involves glucocorticoid replacement to reduce ACTH levels and minimize adrenal androgen production
- in cases of mineralocorticoid deficiency fludrocortisone is also prescribed

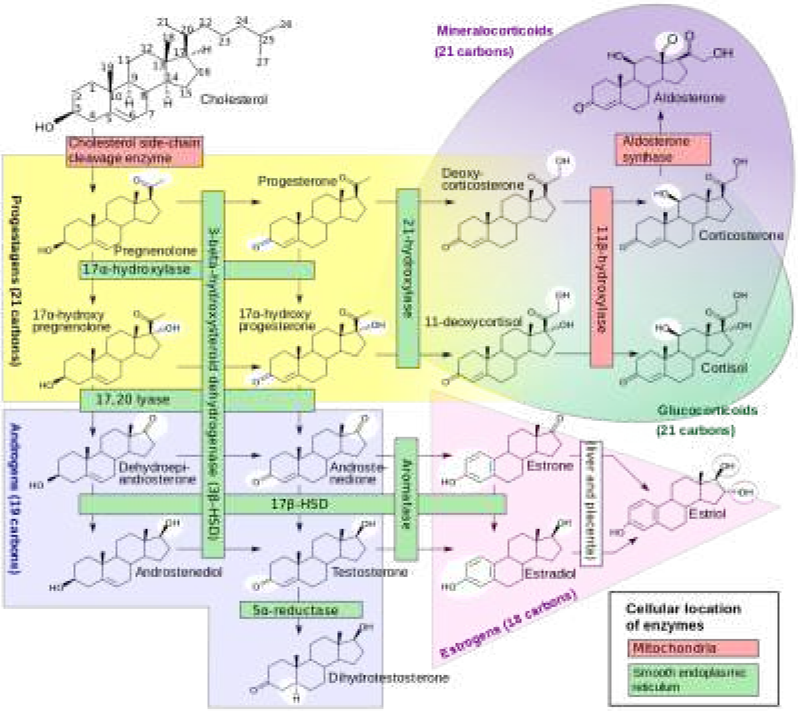

Cholesterol
HO
Cholesterol side-chain cleavage enzyme
Mineralocorticoids (21 carbons)
Aldosterone
Aldosterone synthase
Progestagens (21 carbons)
Pregnenolone
Progesterone
Deoxy-corticosterone
Corticosterone
17α-hydroxylase
3-beta-hydroxysteroid dehydrogenase (3β-HSD)
21-hydroxylase
11β-hydroxylase
17α-hydroxy pregnenolone
17α-hydroxy progesterone
11-deoxycortisol
Cortisol
17,20 lyase
Glucocorticoids (21 carbons)
Androgens (19 carbons)
Dehydroepi-androsterone
Androste-nedione
Aromatase
Estrone
liver and placenta
Estriol
17β-HSD
Androstenediol
Testosterone
Estradiol
5α-reductase
Dihydrotestosterone
Estrogens (18 carbons)
Cellular location of enzymes
Mitochondria
Smooth endoplasmic reticulum

Image sourced from Wikipedia

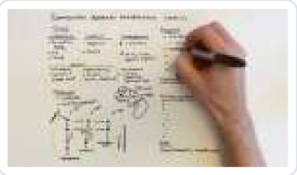
Next question

Textbooks

High-yield textbook

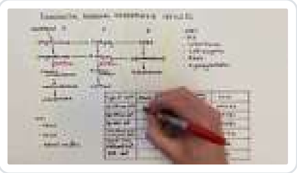
Extended textbook

Media



Congenital Adrenal Hyperplasia (CAH) - 1 of 2

Quick Medical Genetics - YouTube 👍 18 👎 2



Congenital Adrenal Hyperplasia (CAH) - 2 of 2

Quick Medical Genetics - YouTube 👍 13 👎 2

Report broken media

Score: **53.8%**

1	✓
2	✓
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25	✓
26	✓

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Question 27 of 101

A 32-year-old woman presents to her general practitioner with reports of fatigue. She is 30 weeks pregnant with her first child. She has no past medical history and is not on any medications.

Examination reveals a distended abdomen with a symphysis-fundal height that is consistent with gestational age. Foetal movements are noted.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.2 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	145 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Albumin	36 g/L	(35 - 50)

What is the most likely explanation for her symptoms?

○ Acute fatty liver of pregnancy ✕

○ HELLP syndrome ✕

○ Intrahepatic cholestasis of pregnancy ×

○ Normal pregnancy ×

○ Primary biliary cirrhosis ×

Submit answer

Reference ranges ⌵

Score: **53.8%**

1	✓
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Question 27 of 101 ✓

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Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	145 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Albumin	36 g/L	(35 - 50)

What is the most likely explanation for her symptoms?

Acute fatty liver of pregnancy	3%
HELLP syndrome	1%

Intrahepatic cholestasis of pregnancy	14%
Normal pregnancy	82%
Primary biliary cirrhosis	0%

Normal pregnancy can cause a raised ALP - it doesn't necessarily imply liver problems

Important for me Less important

Normal pregnancy is correct. This woman is 30 weeks pregnant and therefore fatigue is not an unexpected complaint. A raised ALP is common in late pregnancy and is usually of placental origin. In the absence of any other signs or symptoms suggesting underlying liver disease, the most likely explanation is that these findings are consistent with normal pregnancy.

HELLP syndrome is incorrect. This stands for haemolysis, elevated liver enzymes and low platelets. She does not have evidence of either haemolysis or low platelets. While fatigue is a possible symptom, typically other symptoms are also present including abdominal pain, nausea, vomiting and backache.

Intrahepatic cholestasis of pregnancy is incorrect. This is typically characterized by itch, which is absent in this case.

Primary biliary cirrhosis is incorrect. While this can cause fatigue and a raised ALP, the median age of diagnosis is 65 and it is relatively uncommon in young people. A placental cause of the raised ALP is far more likely.

Acute fatty liver of pregnancy is incorrect. This is typically characterized by nausea, vomiting and abdominal pain. The ALT level is typically in the 300-500 IU/L range rather than an isolated rise in ALP. The patient will appear unwell, jaundice is common and there may be disseminated intravascular coagulation.

👍 👎 Discuss (2) Improve

Next question

Alkaline phosphatase

Causes of raised alkaline phosphatase (ALP)

- liver: cholestasis, hepatitis, fatty liver, neoplasia
- Paget's
- osteomalacia
- bone metastases
- hyperparathyroidism
- renal failure
- physiological: pregnancy, growing children, healing fractures

The table below splits the causes according to the calcium level

Raised ALP and raised calcium	Raised ALP and low calcium
Bone metastases Hyperparathyroidism	Osteomalacia Renal failure

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.6%**

1	✓
2	✓
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25	✓

26 ✓
27 ✓

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← Question 28 of 101 →

A 62-year-old man presents to hospital with a three day history of tiredness, muscle aches, a fever and pain at the front of his neck. Two weeks ago he had an upper respiratory tract infection which he treated himself with paracetamol and oral decongestants. He did not receive any antibiotics. His only past medical history is mild arthritis of the right knee and he only takes occasional antihistamines during summer time.

On examination, he appears anxious with a fine resting tremor. He has a temperature 38.1°C, a pulse rate of 125 per minute which is regular and normal in character and a blood pressure of 131/78 mmHg. Heart sounds 1 and 2 were present with no added sounds and his chest was clear on auscultation. His abdomen was soft and non-tender with no organomegaly. Neurological examination was unremarkable apart from the resting tremor. Neck examination reveals a diffusely enlarged and tender thyroid gland.

Blood tests are requested and the results are as follows:

Hb	14.0 g/dl	
Platelets	378 * 10^9/l	
WBC	8.9 * 10^9/l	
ESR (Westergren)	94 mm/1st hour	Normal range 0-30
Free T4	214 nmol/l	
Free T3	192 nmol/L	
Plasma TSH	<0.05 mU/l	

Which of the following investigations is likely to be most helpful in establishing the diagnosis?

○ Blood cultures ✕

○ Serum anti-thyroid antibodies ✕

○ Ultrasound scan of neck ✕

○ Radioactive iodine uptake scan ✕

○ Fine needle aspiration ✕

Submit answer

Reference ranges

Score: **55.6%**

1	✓
2	✓
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WBC	8.9 * 10^9/l	
ESR (Westergren)	94 mm/1st hour	Normal range 0-30
Free T4	214 nmol/l	
Free T3	192 nmol/L	
Plasma TSH	<0.05 mU/l	

Which of the following investigations is likely to be most helpful in establishing the diagnosis?

Blood cultures 1%

Serum anti-thyroid antibodies	16%
Ultrasound scan of neck	6%
[✓ Radioactive iodine uptake scan]	75%
Fine needle aspiration	1%

In De Quervain's thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy

Important for me Less important

The patients presenting symptoms and signs are typical of a diagnosis of subacute thyroiditis, of which the most likely cause of this is De Quervains thyroiditis.

Radioactive iodine uptake scan is the most suitable investigation for confirming subacute thyroiditis which demonstrates reduced intake.

👍 👎 Discuss (6) Improve

Next question

Subacute (De Quervain's) thyroiditis ★

Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;

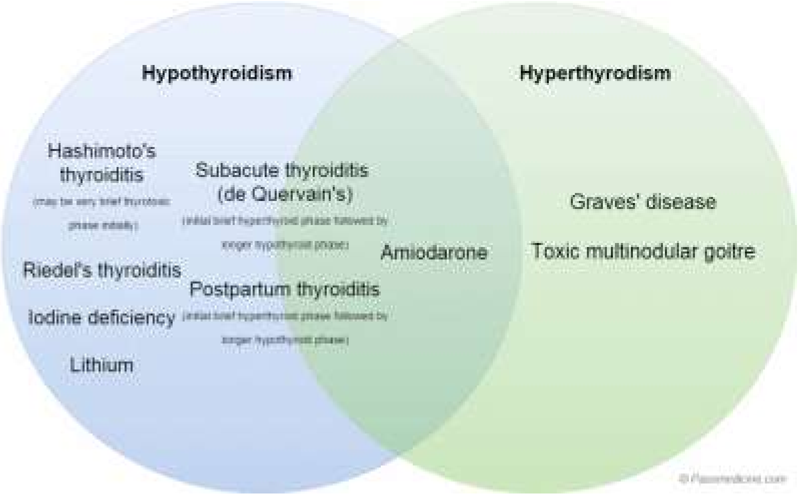
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations

- thyroid scintigraphy: globally reduced uptake of iodine-131

Management

- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Graves' disease
Toxic multinodular goitre
Riedel's thyroiditis
Postpartum thyroiditis
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Iodine deficiency
Lithium
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

+ Q 123

Next question

Textbooks

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Extended textbook

Score: **57.1%**

1	✓
2	✓
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24	✗
25	✓

26 ✓
27 ✓
28 ✓

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Question 29 of 101

A 55 year-old male presents to endocrine outpatient clinic for investigation of gynaecomastia. On examination he has bilateral growth of breast tissue with palpable glandular tissue around the areolae. His past medical history includes hypertension, hypothyroidism, and congestive cardiac failure. He drinks 30 units of alcohol per week. His regular medications include: levothyroxine, amlodipine, bisoprolol, lisinopril and spironolactone.

On examination he is of normal stature, there are no peripheral stigmata of chronic liver disease or testicular masses.

What is the most likely explanation of his gynaecomastia?

- ○ Cirrhosis ×
- ○ Hypopituitarism ×
- ○ Iatrogenic ×
- ○ Klinefelter's syndrome ×
- ○ Idiopathic ×

Submit answer

Reference ranges

Score: **57.1%**

1	✓
2	✓
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7	✗
8	✓
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28	✓
29	-

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← Question 29 of 101 ✓ ⚐ →

A 55 year-old male presents to endocrine outpatient clinic for investigation of gynaecomastia. On examination he has bilateral growth of breast tissue with palpable glandular tissue around the areolae. His past medical history includes hypertension, hypothyroidism, and congestive cardiac failure. He drinks 30 units of alcohol per week. His regular medications include: levothyroxine, amlodipine, bisoprolol, lisinopril and spironolactone.

On examination he is of normal stature, there are no peripheral stigmata of chronic liver disease or testicular masses.

What is the most likely explanation of his gynaecomastia?

Cirrhosis	5%
Hypopituitarism	3%
Iatrogenic	88%
Klinefelter's syndrome	1%
Idiopathic	3%

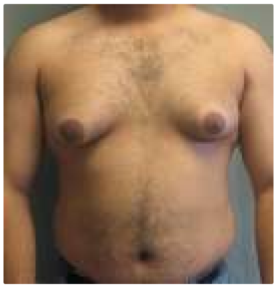
The most likely explanation for this gentlemans gynaecomastia given the negative examination findings is a side effect of his current regular medications. There are many drugs that can cause gynaecomastia; in this cause the culprit could be either amlodipine or spironolactone.

Discuss (4) Improve

Next question

Gynaecomastia

Gynaecomastia describes an abnormal amount of breast tissue in males and is usually caused by an increased oestrogen:androgen ratio. It is important to differentiate the causes of galactorrhoea (due to the actions of prolactin on breast tissue) from those of gynaecomastia



Causes of gynaecomastia

- physiological: normal in puberty
- syndromes with androgen deficiency: Kallman's, Klinefelter's
- testicular failure: e.g. mumps
- liver disease
- testicular cancer e.g. seminoma secreting hCG
- ectopic tumour secretion
- hyperthyroidism
- haemodialysis
- drugs: see below

Drug causes of gynaecomastia

- spironolactone (most common drug cause)
- cimetidine
- digoxin
- cannabis
- finasteride
- GnRH agonists e.g. goserelin, buserelin
- oestrogens, anabolic steroids

Very rare drug causes of gynaecomastia

- tricyclics
- isoniazid

- calcium channel blockers
- heroin
- busulfan
- methyldopa

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Dr. Jamil Alrajhi

Score: **58.6%**

1	✓
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Question 30 of 101

A 19-year-old man is reviewed prior to discharge. He presented with vomiting and abdominal pain and was found to have diabetic ketoacidosis. He was managed as an inpatient for five days before being well enough for discharge. He is also diagnosed with type 1 diabetes mellitus on this admission as a cause of the diabetic ketoacidosis. He has been educated by the diabetic nurse on how to manage his diabetes and insulin at home, but he is concerned about what his target plasma glucose should be after eating.

What is the recommended target after eating to be achieved by home monitoring?

○ 3-6mmol/litre ×

○ 5-10mmol/litre ×

○ 5-9mmol/litre ×

○ 7-12mmol/litre ×

○ 2-9 mmol/litre ×

Submit answer

Reference ranges

Score: **58.6%**

1 ✓

2	✓
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What is the recommended target after eating to be achieved by home monitoring?

🟥	3-6mmol/litre	6%
	5-10mmol/litre	20%
🟩	5-9mmol/litre	46%
	7-12mmol/litre	27%
	2-9 mmol/litre	0%

The correct answer is 5-9mmol/litre. NICE recommends that people with type 1 diabetes should aim for 5-7mmol/litre on waking, 4-7mmol/litre before meals and 5-9mmol/litre 90 minutes after eating. Frequent testing is very important in patients starting with insulin therapy to avoid both high and low sugar levels.

Source:

'Type 1 diabetes in adults: diagnosis and management' Clinical guideline [NG17]. The National Institute for Health and Care Excellence, August 2015.

👍 👎 Discuss (1) Improve

Next question

Insulin therapy

Insulin therapy revolutionised the management of diabetes mellitus when it was developed in the 1920's. It is still the only available treatment for type 1 diabetes mellitus (T1DM) and is widely used in type 2 diabetes mellitus (T2DM) where oral hypoglycaemic agents fail to gain adequate control.

It can sometimes seem daunting to understand the various types of insulin but it is important you have a basic grasp to avoid potential harm to patients.

Classification of insulin

By manufacturing process
- porcine: extracted and purified from pig pancreas
- human sequence insulin: either produced by enzyme modification of porcine insulin (emp) or biosynthetically by recombinant DNA using bacteria (crb, prb) or yeast (pyr)
- analogues

By duration of action

	Onset	**Peak**	**Duration**
Rapid-acting insulin analogues	5 mins	1 hour	3-5 hours
Short-acting insulin	30 mins	3 hours	6-8 hours
Intermediate-acting insulin	2 hours	5-8 hours	12-18 hours

	Onset	Peak	Duration
Long-acting insulin analogues	1-2 hours	Flat profile	Up to 24 hours
Premixed preparations	-	-	-

Patients often require a mixture of preparations (e.g. both short and long acting) to ensure stable glycaemic control throughout the day.

Rapid-acting insulin analogues
- the rapid-acting human insulin analogues act faster and have a shorter duration of action than soluble insulin (see below)
- may be used as the bolus dose in 'basal-bolus' regimes (rapid/short-acting 'bolus' insulin before meals with intermediate/long-acting 'basal' insulin once or twice daily)
- insulin aspart: NovoRapid
- insulin lispro: Humalog

Short-acting insulins
- soluble insulin examples: Actrapid (human, pyr), Humulin S (human, prb)
- may be used as the bolus dose in 'basal-bolus' regimes

Intermidate-acting insulins
- isophane insulin
- many patients use isophane insulin in a premixed formulation with

Long-acting insulins
- insulin determir (Levemir): given once or twice daily
- insulin glargine (Lantus): given once daily

Premixed preparations
- combine intermediate acting insulin with either a rapid-acting insulin analogue or soluble insulin
- Novomix 30: 30% insulin aspart (rapid-acting), 70% insulin aspart protamine (intermediate-acting)

- Humalog Mix25: 25% insulin lispro (rapid-acting), 75% insulin lispro protamine (intermediate-acting); Humalog Mix50: 50% insulin lispro, 50% insulin lispro protamine
- Humulin M3: biphasic isophane insulin (human, prb) - 30% soluble (short-acting), 70% isophane (intermediate-acting)
- Insuman Comb 15: biphasic isophane insulin 9human, prb) - 30% soluble (short-acting), 70% isophane (intermediate-acting)

Administration of insulin

The vast majority of patients administer insulin subcutaneously. It is important to rotate injection sites to prevent lipodystrophy. Insulin pumps are available ('continuous subcutaneous insulin infusions') which delivers a continuous basal infusion and a patient-activated bolus dose at meal times.

Intravenous insulin is used for patients who are acutely unwell, for example with diabetic ketoacidosis. Inhaled insulin is available but not widely used and oral insulin analogues are in development but have considerable technical hurdles to clear.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **56.7%**

1	✓
2	✓
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30 ✗

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A 28-year-old woman attends your pre-natal endocrinology clinic. She has been taking 100 micrograms of levothyroxine daily due to hypothyroidism and is currently 6 weeks pregnant, there have been no recent changes to her dosage.

Thyroid stimulating hormone (TSH)	5.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	10.0 pmol/L	(9.0 - 18)

What would you recommend regarding her levothyroxine dose?

- ○ Continue on current levothyroxine dose, no further monitoring required during pregnancy
- ○ Increase levothyroxine dose to 150 micrograms daily
- ○ Increase levothyroxine dose to 200 micrograms once daily immediately
- ○ Reduce levothyroxine dose to 75 micrograms once daily
- ○ Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose

Submit answer

Reference ranges

Score: **56.7%**

1	✓
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Free thyroxine (T4)	10.0 pmol/L	(9.0 - 18)

What would you recommend regarding her levothyroxine dose?

Continue on current levothyroxine dose, no further monitoring required during pregnancy	0%
Increase levothyroxine dose to 150 micrograms daily	91%
Increase levothyroxine dose to 200 micrograms once daily immediately	2%
Reduce levothyroxine dose to 75 micrograms once daily	1%
Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose	5%

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

Important for me Less important

During pregnancy, the thyroid gland increases in size by 10% and the production of thyroid hormones, T3 and T4, increases by 50%.

If a patient has pre-existing hypothyroidism, we usually need to increase the dose of levothyroxine to mimic these changes.

Increase levothyroxine dose to 150 micrograms daily: an increase of levothyroxine supplementation of up to 50% mimics the physiological thyroid response to pregnancy and is the correct option for this patient.

Continue on current levothyroxine dose, no further monitoring required during pregnancy: this is incorrect. The requirement for T3 and T4 increases during pregnancy, therefore the levothyroxine dose must be increased.

Increase levothyroxine dose to 200 micrograms once daily immediately: this increase is too high, the patient does not require such an aggressive dosage adjustment.

Reduce levothyroxine dose to 75 micrograms once daily: incorrect, levothyroxine dose should not routinely be reduced during pregnancy, the requirement generally increases.

Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose: this would be inappropriate, thyroid abnormalities should be treated as soon as possible to minimise the risk to the foetus. Ideally, the patient should be counselled before conception. Maternal hypothyroidism is associated with low birth weight, premature labour, and neonatal respiratory problems.

👍 👎 Discuss (1) Improve

Next question

Pregnancy: thyroid problems ★

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points
- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
  - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link

Report broken link

Score: **58.1%**

1	✓
2	✓
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27	✓
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Question 32 of 101

A 62-year-old patient presents to review in the diabetes clinic. He has had type 2 diabetes for 10 years, as well as previous appendectomy, kidney stones, previous tibial fracture and depression which is now in remission. He takes metformin and gliclazide only and has no allergies. His HbA1c is 40mmol/mol, urine dip is negative for glucose and protein but his blood pressure is 151/96mmHg. He has previously attempted to control his blood pressure by diet and exercise only. He is a white Caucasian. What is the most appropriate plan in regards to blood pressure management?

- ○ Start a beta-blocker ×
- ○ Start a thiazide-like diuretic ×
- ○ Start an ACE inhibitor ×
- ○ Start an angiotensin II receptor blocker ×
- ○ Start a calcium channel blocker ×

Submit answer

Reference ranges

Score: **58.1%**

1 ✓
2 ✓
3 ✗

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Question 32 of 101 ✓

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Start a beta-blocker	0%
Start a thiazide-like diuretic	2%
Start an ACE inhibitor	76%
Start an angiotensin II receptor blocker	6%
Start a calcium channel blocker	15%

A patient with T2DM with a new diagnosis of hypertension should be treated with an ACE inhibitor as first line treatment, regardless of age

Important for me Less important

The correct answer is to start a ACE inhibitor. This is because he has diabetes. A patient with diabetes should have an ACE inhibitor as first line treatment for diabetes regardless of their age. If the patient did not have diabetes, then a calcium channel blocker would be the most appropriate option. If the patient could not tolerate a ACE inhibitor then an angiotensin II receptor blocker should be tried. ACE inhibitors should also start for patients with diabetes if there is proteinuria, and therefore should have at least annual urinary albumin to creatinine ratios measured. Beta-blockers are only used after ACE inhibitors, calcium channel blockers, diuretics and alpha blockers have failed to controlled blood pressure. However, IV labetalol can be used in hypertensive emergencies to reduce blood pressure.

👍 👎 Discuss (8) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

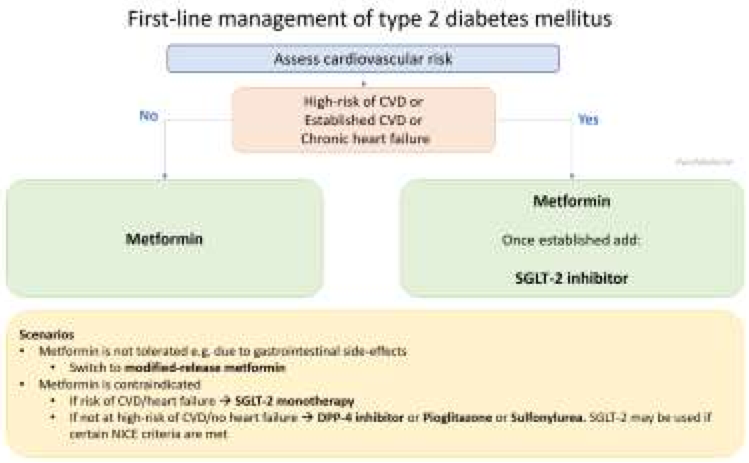
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
    - Switch to **modified-release metformin**
- Metformin is contraindicated
    - If risk of CVD/heart failure → **SGLT-2 monotherapy**
    - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

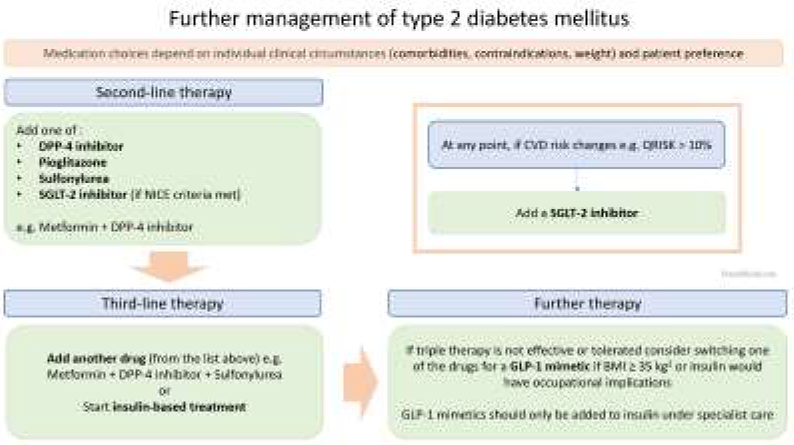
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a

QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone

- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

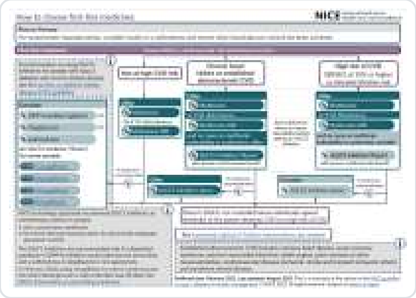
GLP-1 mimetics should only be added to insulin under specialist care

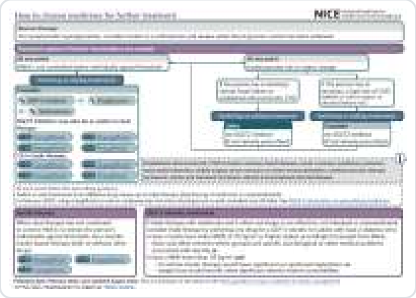
Starting insulin

- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

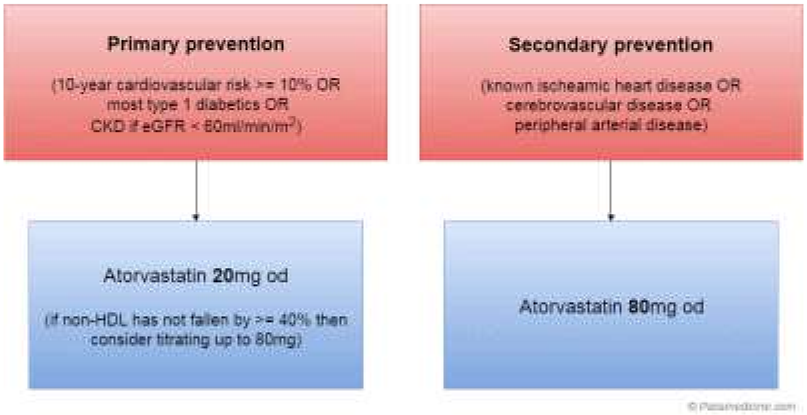
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 33 40

2014 Lipid modification guidelines

SIGN 36 41

2010 Management of diabetes

NICE 👍 56 👎 48

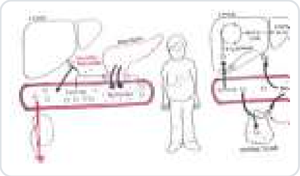
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

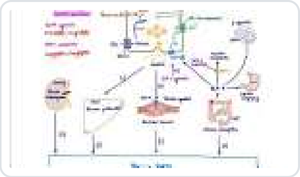
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **59.4%**

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32 ✓

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A 68-year-old gentleman was admitted to the medical admissions unit with increasing drowsiness. He had been diagnosed with primary small cell carcinoma of the lung six months ago and had declined curative chemotherapy. At the time of diagnosis there was no evidence of metastasis, and his past medical history comprised of chronic obstructive pulmonary disease, ischaemic heart disease, hypertension, hypercholesterolaemia and depression.

His wife had taken him to the Emergency Department having noted that he had been increasingly drowsy over the last few hours, as well as developing new onset confusion. He had otherwise been relatively well prior to the admission. He had consulted his GP about new onset generalised aches and pains within the last four weeks for which his GP had commenced Oramorph solution PRN. Since then he had developed abdominal pain which the GP had diagnosed as secondary to opiate-induced constipation and he was accordingly prescribed lactulose 15ml BD with partial relief of his symptoms. There had been no evidence of weakness or numbness and no evidence of speech impairment; as far as his wife was aware he had taken the prescribed dose of oramorph. His drug history comprised of oramorph solution 10mg BD, paracetamol 1g QDS, dihydrocodeine 60mg QDS, lactulose 15ml BD, aspirin 75mg OD, atorvastatin 20mg ON, bisoprolol 2.5mg OD, Ramipril 2.5mg OD and furosemide 40mg OD.

Examination revealed a drowsy gentleman with a GCS of 12 (E 3 M5 V4). His blood pressure was 102/68, heart rate 58bpm, respiratory rate 10/min, oxygen saturations of 95% on air and temperature 36.6ºC. Examination of his cardiovascular and respiratory systems were unremarkable. Examination of his central nervous system revealed the presence of normal sized pupils; he was not compliant with formal neurological examination but no other focal neurological signs were found. There was no evidence of neck stiffness and Kernig's sign was negative. He was not cooperative with an abbreviated mental state examination.

Which investigation is most likely to be diagnostic of the underlying cause?

- ○ Urgent CT head scan ×
- ● Urgent serum liver function and calcium profile ×
- ○ Urgent isotope bone scan ×
- ○ Urgent septic screen ×
- ○ Urgent PET scan ×

Submit answer

Reference ranges

Score: **59.4%**

1	✓
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Which investigation is most likely to be diagnostic of the underlying cause?

Urgent CT head scan	24%
Urgent serum liver function and calcium profile	61%
Urgent isotope bone scan	4%
Urgent septic screen	2%
Urgent PET scan	8%

This gentleman has developed signs of life-threatening hypercalcaemia, having manifested previous potential symptoms including new onset abdominal pain and constipation. This question is asking what the single next most important investigation should be, and whilst most of the above options are relevant, candidates are asked to discriminate from what is deemed to be the next essential management should be. It is likely that he has developed metastasis to his spine thus whilst an isotope bone scan may be an important part of his overall management, it is not as relevant in the acute setting. Likewise, it is likely that a septic screen and CT head would be performed, but clinically speaking there is little definite evidence of acute raised intracranial pressure or sepsis. There is no indication at present that he is suffering from opiate toxicity and therefore IV naloxone should not be administered at this stage.

👍 👎 Discuss (20) Improve

Next question

Hypercalcaemia: causes ★

Two conditions account for 90% of cases of hypercalcaemia:

- 1. Primary hyperparathyroidism: commonest cause in non-hospitalised patients

- 2. Malignancy: the commonest cause in hospitalised patients. This may be due to a number of processes, including;
  - PTHrP from the tumour e.g. squamous cell lung cancer
  - bone metastases
  - myeloma,: due primarily to increased osteoclastic bone resorption caused by local cytokines (e.g. IL-1, tumour necrosis factor) released by the myeloma cells
- for this reason, measuring parathyroid hormone levels is the key investigation for patients with hypercalcaemia

Other causes include

- sarcoidosis
  - other causes of granulomas may lead to hypercalcaemia e.g. tuberculosis and histoplasmosis
- vitamin D intoxication
- acromegaly
- thyrotoxicosis
- Milk-alkali syndrome
- drugs:
  - thiazides
  - calcium-containing antacids
- dehydration
- Addison's disease
- Paget's disease of the bone
  - usually normal in this condition but hypercalcaemia may occur with prolonged immobilisation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 20 👎 8

Hypercalcaemia â€“ presentation and management

Suggest link Report broken link

Media

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 4 👎 0

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

Report broken media

Score: **60.6%**

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Question 34 of 101

A 65-year-old man presents is referred to the urology clinic with three weeks of macroscopic haematuria. He has a past medical history of type 2 diabetes. He recently underwent treatment for a threadworm infection. His medications include linagliptin, metformin and pioglitazone. He does not smoke cigarettes or drink alcohol. He works as a dairy farmer. He is originally from Jamaica.

He undergoes a cystoscopy, which demonstrates an exophytic lesion in the bladder.

Given the likely diagnosis, what is the patient's most significant risk factor for the development of this condition?

- ○ Ethnicity ×
- ○ Threadworm infection ×
- ○ Metformin ×
- ○ Occupation ×
- ○ Pioglitazone ×

Submit answer

Reference ranges

Score: **60.6%**

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Question 34 of 101

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He undergoes a cystoscopy, which demonstrates an exophytic lesion in the bladder.

Given the likely diagnosis, what is the patient's most significant risk factor for the development of this condition?

○ Ethnicity ✕

○ Threadworm infection ✕

○ Metformin ✕

○ Occupation ✕

○ Pioglitazone ✕

Submit answer

Reference ranges

Score: **60.6%**

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A 65-year-old man presents is referred to the urology clinic with three weeks of macroscopic haematuria. He has a past medical history of type 2 diabetes. He recently underwent treatment for a threadworm infection. His medications include linagliptin, metformin and pioglitazone. He does not smoke cigarettes or drink alcohol. He works as a dairy farmer. He is originally from Jamaica.

He undergoes a cystoscopy, which demonstrates an exophytic lesion in the bladder.

Given the likely diagnosis, what is the patient's most significant risk factor for the development of this condition?

Ethnicity	2%
Threadworm infection	12%
Metformin	1%
Occupation	7%
Pioglitazone	78%

Thiazolidinediones are associated with an increased risk of bladder cancer

Important for me Less important

Pioglitazone is correct. Macroscopic haematuria and an exophytic lesion identified on cystoscopy suggest bladder cancer. Thiazolidinediones are a well-recognized risk factor for the development of bladder cancer.

Ethnicity is incorrect. Bladder cancer more commonly develops in those of white ethnicity rather than black. However, black patients are twice as likely to die from bladder cancer.

Occupation is incorrect. Typically, those employed in industries such as textile, rubber, leather, dye, paint, and print are at increased risk of bladder cancer due to chemical exposures. Dairy farming is not a typical risk factor.

Metformin is incorrect. The use of this medication is not associated with the development of bladder cancer.

Threadworm infection is incorrect. This parasite is not associated with bladder cancer. The parasitic infection associated with the development of bladder cancer is schistosomiasis. This is endemic to various tropical and subtropical regions. It is not typically found in Jamaica.

👍 👎 💬 Discuss (3) Improve

Next question

Thiazolidinediones ★

Thiazolidinediones are a class of agents used in the treatment of type 2 diabetes mellitus. They are agonists to the PPAR-gamma receptor and reduce peripheral insulin resistance. Rosiglitazone was withdrawn in 2010 following concerns about the cardiovascular side-effect profile.

The PPAR-gamma receptor is an intracellular nuclear receptor. It's natural ligands are free fatty acids and it is thought to control adipocyte differentiation and function.

Adverse effects

- weight gain
- liver impairment: monitor LFTs
- fluid retention - therefore contraindicated in heart failure. The risk of fluid retention is increased if the patient also takes insulin
- recent studies have indicated an increased risk of fractures
- bladder cancer: recent studies have shown an increased risk of bladder cancer in patients taking pioglitazone (hazard ratio 2.64)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 11 👎 7

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Score: **61.8%**

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A 56-year-old lady presents with a 3 month history of abdominal pains, low mood and constipation. Past medical history includes hypertension and depression following the death of her husband 2 years ago. Routine blood tests are performed by the GP and upon review the patient is referred into hospital.

Blood tests are as below:

Hb	100 g/l	Na$^+$	135 mmol/l
Platelets	230 * 10^9/l	K$^+$	4.7 mmol/l
WBC	10 * 10^9/l	Urea	6 mmol/l
Calcium (adjusted)	2.96 mmol/l	Creatinine	110 μmol/l
Phosphate	1.35 mmol/l	CRP	30 mg/l
Albumin	35 g/L		

Which diagnostic test should be performed first?

○ Parathyroid hormone level ×

○ Myeloma screen ×

○ CT chest, abdomen and pelvis ×

○ Urinary calcium levels ×

○ Skeletal X-ray ×

Submit answer

Reference ranges

Score: **61.8%**

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WBC	10 * 10^9/l	Urea	6 mmol/l
Calcium (adjusted)	2.96 mmol/l	Creatinine	110 µmol/l
Phosphate	1.35 mmol/l	CRP	30 mg/l
Albumin	35 g/L		

Which diagnostic test should be performed first?

Parathyroid hormone level	83%
Myeloma screen	9%
CT chest, abdomen and pelvis	3%
Urinary calcium levels	4%
Skeletal X-ray	1%

The two main causes of hypercalcaemia are primary hyperparathyroidism and malignancy. Parathyroid hormone level will help to differentiate between these two main differentials and help guide further investigations.

👍 👎 Discuss (11) Improve

Next question

Hypercalcaemia: causes

Two conditions account for 90% of cases of hypercalcaemia:

- 1. Primary hyperparathyroidism: commonest cause in non-hospitalised patients
- 2. Malignancy: the commonest cause in hospitalised patients. This may be due to a number of processes, including;
  - PTHrP from the tumour e.g. squamous cell lung cancer
  - bone metastases
  - myeloma,: due primarily to increased osteoclastic bone resorption caused by local cytokines (e.g. IL-1, tumour necrosis factor) released by the myeloma cells
- for this reason, measuring parathyroid hormone levels is the key investigation for patients with hypercalcaemia

Other causes include

- sarcoidosis
  - other causes of granulomas may lead to hypercalcaemia e.g. tuberculosis and histoplasmosis
- vitamin D intoxication
- acromegaly
- thyrotoxicosis
- Milk-alkali syndrome
- drugs:
  - thiazides
  - calcium-containing antacids
- dehydration
- Addison's disease
- Paget's disease of the bone
  - usually normal in this condition but hypercalcaemia may occur with prolonged immobilisation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 👍 20 👎 8

Hypercalcaemia â€“ presentation and management

Suggest link Report broken link

Media

HYPERCALCEMIA

Hypercalcemia

Osmosis - YouTube 👍 4 👎 0

ONCOLOGY
HYPERCALCAEMIA

Hypercalcemia in malignancy

Armando Hasudungan - YouTube 👍 1 👎 0

Report broken media

Score: **62.9%**

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Question 36 of 101

A 35-year-old woman is known to have a family history of multiple endocrine neoplasia type 2a. She has tested positive for the RET oncogene and is concerned as her mother had suffered from thyroid cancer. However, she is reluctant to undergo a prophylactic thyroidectomy and asked if there is another option available.

Which of the following management options would be appropriate for her case?

- ○ Annual monitoring of thyroid peroxidase (TPO) ×
- ○ Annual monitoring of thyroglobulin ×
- ○ Annual 5 hydroxyindoleacetic acid levels (5-HIAA) ×
- ○ Annual monitoring of calcitonin ×
- ○ Annual monitoring of chromogranin A ×

Submit answer

Reference ranges

Score: **62.9%**

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31	✓
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33	✓
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36	-

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Which of the following management options would be appropriate for her case?

	Annual monitoring of thyroid peroxidase (TPO)	4%
	Annual monitoring of thyroglobulin	20%
	Annual 5 hydroxyindoleacetic acid levels (5-HIAA)	2%
✓	Annual monitoring of calcitonin	73%
	Annual monitoring of chromogranin A	2%

Calcitonin can be used as a marker to monitor for Medullary thyroid carcinoma in MEN type II in those unwilling to undergo prophylactic thyroidectomy

Important for me Less important

Calcitonin is a marker for medullary thyroid carcinoma which is associated with MEN type II.

Chromogranin A and 5-HIAA can be used as markers for neuroendocrine tumours and carcinoid syndrome respectively.

Thyroid peroxidase antibody titre can be used as a marker in autoimmune thyroid conditions such as Hashimoto's thyroiditis and Graves disease.

Thyroglobulin can be used as a marker for papillary or follicular thyroid carcinoma. For example, a rise in this marker following a thyroidectomy can indicate the recurrence of these particular cancers.

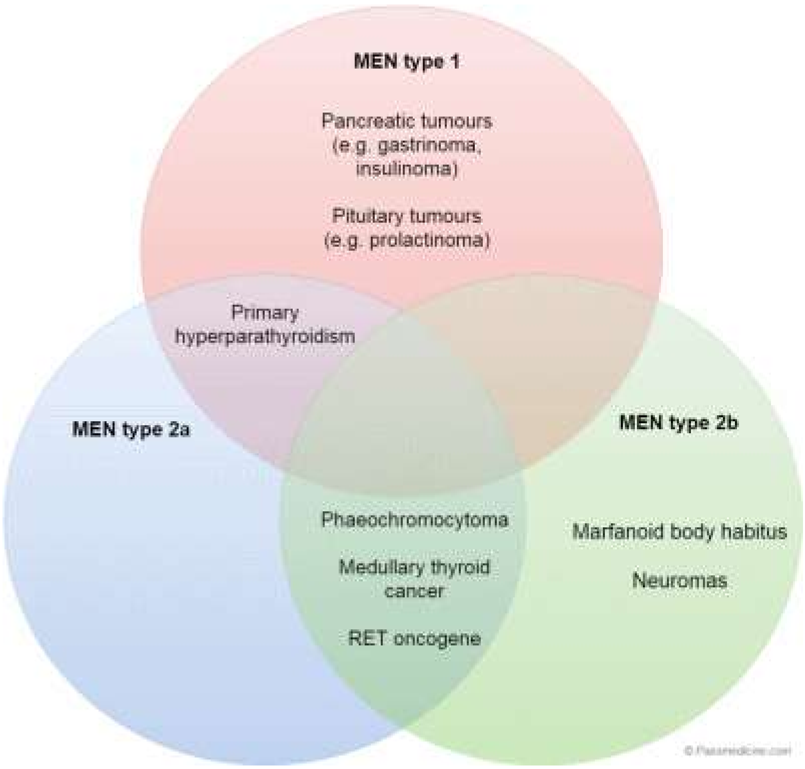
👍 👎 Discuss (4) Improve

Next question

Multiple endocrine neoplasia

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
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Venn diagram showing the different types of MEN and their associated features

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Pancreatic
Neuroendocrine
Neoplasms

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **63.9%**

1 ✓
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You are the medical doctor on an acute medical unit. A 28 year old, adopted female with a history of recurrent kidney stones has been referred by her GP with hypokalaemia (K+ 2.6 mmol/l). She feels well in herself and denies any vomiting or diarrhoea. Her observations are normal and blood results are as below:

Na$^+$	141 mmol/l
K$^+$	2.6 mmol/l
Bicarbonate	18 mmol/l
Urea	5.0 mmol/l
Creatinine	67 µmol/l
Anion gap	13 mEq/L

What is the most likely cause of her presentation?

- ○ Liquorice poisoning ×
- ○ Renal tubular acidosis type 1 ×
- ○ Renal tubular acidosis type 2 ×
- ○ Renal tubular acidosis type 3 ×
- ○ Renal tubular acidosis type 4 ×

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **63.9%**

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K^+	2.6 mmol/l
Bicarbonate	18 mmol/l
Urea	5.0 mmol/l
Creatinine	67 µmol/l
Anion gap	13 mEq/L

What is the most likely cause of her presentation?

Liquorice poisoning	1%
[✓ Renal tubular acidosis type 1]	81%
Renal tubular acidosis type 2	13%
Renal tubular acidosis type 3	1%
Renal tubular acidosis type 4	4%

The patient has presented with a history of recurrent kidney stones, hypokalaemia, acidosis and a normal anion gap. This is typical for renal tubular acidosis type 1. Renal tubular acidosis type 2 would present with similar biochemical features but is more unlikely to have a history of kidney stones. Renal tubular acidosis type 3 is an obsolete concept. Renal tubular acidosis type 4 is associated with hyperkalaemia.

Liquorice poisoning would cause hypokalaemia and hypernatraemia but is not associated with the other biochemical abnormalities found here.

👍 👎 Discuss (6) Improve

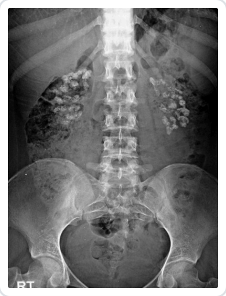
Next question

Renal tubular acidosis

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare

- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

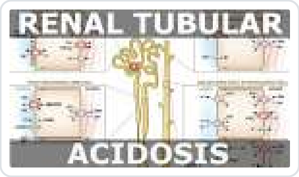
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 8 0


RENAL TUBULAR
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Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **64.9%**

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Question 38 of 101

A 37-year-old man presents to the endocrinology clinic for review. He was started on hydrocortisone for Addison's disease six months ago following a history of feeling generally unwell, postural dizziness and vomiting. Following starting hydrocortisone he felt much better. He was originally started on 10mg in the morning and 5mg at lunchtime and 5mg in the early afternoon. This was further increased to 10mg, 10mg and 5mg for the morning, lunchtime, and afternoon, respectively. The patient feels that all of the original symptoms have resolved, but he has noticed swelling of his ankles. On examination, he has non-pitting oedema of his legs. He has a heart rate of 78bpm, blood pressure of 165/102mmHg, saturations of 98% on room air and a respiratory rate of 14 breaths per minute. His previous blood pressure recorded in clinic had been 105/61mmHg He has no other medical problems, and takes no other tablets apart from paracetamol and ibuprofen when needed, especially for headaches. What is the most appropriate action?

- ○ Start amlodipine ×
- ○ Start ACE inhibitor ×
- ○ Reduce dose of hydrocortisone ×
- ○ Increase dose of hydrocortisone ×
- ○ Start beta-blocker ×

Submit answer

Reference ranges

Score: **64.9%**

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Question 38 of 101 ✓

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Start amlodipine	3%
Start ACE inhibitor	24%
Reduce dose of hydrocortisone	67%
Increase dose of hydrocortisone	5%
Start beta-blocker	2%

Features of over-replacement of corticosteroids require dose adjustment

Important for me Less important

This is a patient on treatment for Addison's disease who has developed evidence of over-replacement. These features can be any of Cushing's disease; such as hypertension for this patient. The increase in blood pressure with steroids implies that this is not essential hypertension but rather secondary hypertension. This implies that he is having a too much steroid replacement and hydrocortisone dose should be reduced. Features of over-replacement include hypertension, thin skin, striae, easy bruising, hyperglycaemia and electrolyte abnormalities. Features of under-replacement include fatigue, postural hypotension, weight loss and salt craving.

👍 👎 💬 Discuss (8) Improve

Next question

Addison's disease: management ★

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:
- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:
- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards
- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 16

Addison's disease

Royal College of Physicians 👍 16 👎 8

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube

👍 1 👎 0

[Primary adrenal insufficiency (Addison's disease)]

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **65.8%**

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38	✓

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← Question 39 of 101 →

A 58-year-old patient who has a history of hypertension is operated on by the neurosurgeons for an intracranial haemorrhage.

Over the next few days his serum sodium level progressively declines and by the third day has fallen to 118 mmol/l despite fluid restriction to 1L per day. Urine osmolarity is 700 mOsmo/l and urinary sodium is raised at 80 mmol/l.

What is the most likely diagnosis?

- ○ Addisonian crisis ✕
- ○ Secretion of inappropriate antidiuretic hormone ✕
- ○ Cranial diabetes insipidus ✕
- ○ Hypovolaemia ✕
- ○ Fluid overload ✕

Submit answer

Reference ranges

Score: **65.8%**

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39 -

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Question 39 of 101 ✓

A 58-year-old patient who has a history of hypertension is operated on by the neurosurgeons for an intracranial haemorrhage.

Over the next few days his serum sodium level progressively declines and by the third day has fallen to 118 mmol/l despite fluid restriction to 1L per day. Urine osmolarity is 700 mOsmo/l and urinary sodium is raised at 80 mmol/l.

What is the most likely diagnosis?

Addisonian crisis	3%
Secretion of inappropriate antidiuretic hormone	80%
Cranial diabetes insipidus	15%
Hypovolaemia	2%
Fluid overload	1%

The hyponatraemia and hypotonic blood plasma, coupled with the raised urine osmolality and raised urinary sodium excretion indicates a diagnosis of syndrome of inappropriate ADH secretion. This is a condition that can occur after head trauma, a central nervous system infection and intracranial haemorrhage.

Discuss (7) | Improve

Next question

Syndrome of inappropriate ADH secretion

The syndrome of inappropriate ADH secretion (SIADH) is characterised by hyponatraemia secondary to the dilutional effects of excessive water retention. Patients with SIADH are typically euvolemic, i.e. they do not show signs of overt volume depletion or overload.

Pathophysiology

- SIADH involves an excessive release of antidiuretic hormone (ADH), also known as vasopressin, which leads to water retention, volume expansion, and dilutional hyponatraemia
- ADH is produced by the hypothalamus and stored in the posterior pituitary gland. Its primary function is to regulate the body's water balance
- It does this by increasing water reabsorption in the collecting ducts of the kidneys, thereby decreasing the volume of urine produced
- In SIADH, there is an inappropriate and continuous release of ADH that is not inhibited by normal physiological mechanisms, such as adequate or excess body fluid levels
- As a result, the kidneys reabsorb more water, leading to decreased urine output, and expansion of extracellular fluid volume.
- Importantly, this increase in body fluid volume does not lead to the expected signs of fluid overload, such as oedema or hypertension, because the excess fluid is uniformly distributed throughout all body fluid compartments.
- However, as water is retained in the body, the concentration of electrolytes in the blood, particularly sodium, becomes diluted, leading to hyponatraemia.

Causes of SIADH

Category	Examples
Malignancy	• **small cell lung cancer** • also: pancreas, prostate
Neurological	• stroke • subarachnoid haemorrhage • subdural haemorrhage • meningitis/encephalitis/abscess

Category	Examples
Infections	• tuberculosis • pneumonia
Drugs	• sulfonylureas* • SSRIs, tricyclics • carbamazepine • vincristine • cyclophosphamide
Other causes	• positive end-expiratory pressure (PEEP) • porphyrias

Investigations

- Urine osmolality: Urine osmolality is inappropriately high (>100 mOsm/kg) in relation to serum osmolality, as the kidneys should normally dilute urine in the setting of low serum osmolality.
- Urine sodium concentration: Urine sodium concentration is typically high (>40 mmol/L) due to the action of ADH on the renal tubules.

Management

- correction must be done slowly to avoid precipitating central pontine myelinolysis
- fluid restriction
- demeclocycline: reduces the responsiveness of the collecting tubule cells to ADH
- ADH (vasopressin) receptor antagonists have been developed

*the BNF states this has been reported with glimepiride and glipizide.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

WHAT IS...
SIADH?

Syndrome of inappropriate antidiuretic hormone (SIADH)

Osmosis - YouTube 👍 11 👎 2

Report broken media

Score: **66.7%**

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Question 40 of 101

A 65-year-woman is referred to the endocrinology department by her general practitioner. She complains of 12 months of enlarging hands and feet with an inability to put on her usual rings. She also reports that her facial appearance has changed and complains of persistent sweating and arthralgia. She has no significant past medical history and is on no regular medications.

On examination, it is noted that she has coarsened facial features and frontal bossing. There is macroglossia. Neurological examination reveals bitemporal hemianopia. Her hands and feet appear large and spade-like. There was no synovitis.

Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	6.2 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
IGF-1	38.2 nmol/L	(7.6-23.1)

An MRI head reveals a large pituitary macroadenoma.

An oral glucose tolerance test is arranged and there is a failure to suppress growth hormone with an oral glucose load.

The patient undergoes trans-sphenoidal surgery to remove the tumour.

Despite this intervention, she complains of ongoing sweating, arthralgia and fatigue.

A repeat MRI demonstrates a small amount of residual tumour that is deemed inaccessible to further surgical intervention.

A repeat IGF-1 remains elevated (35 nmol/L) and there is persistent failure to suppress growth hormone with an oral glucose load.

Given the likely diagnosis, what is the most appropriate management at this point?

- ○ Bromocriptine ✕
- ○ Cabergoline ✕
- ○ Octreotide ✕
- ○ Pegvisomant ✕
- ○ Radiotherapy ✕

Submit answer

Reference ranges ∨

Score: **66.7%**

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A repeat MRI demonstrates a small amount of residual tumour that is deemed inaccessible to further surgical intervention.

A repeat IGF-1 remains elevated (35 nmol/L) and there is persistent failure to suppress growth hormone with an oral glucose load.

Given the likely diagnosis, what is the most appropriate management at this point?

Bromocriptine	9%
Cabergoline	7%
[✓ Octreotide]	57%
Pegvisomant	17%
Radiotherapy	11%

Acromegaly: if patients are not suitable for trans-sphenoidal surgery, or have residual symptoms, then octreotide may be used

Important for me Less important

Octreotide is the correct answer. This patient has acromegaly, which is the consequence of a growth hormone-secreting pituitary tumour. Clinical features include enlargement of hands and feet, coarsening of facial features and frontal bossing. IGF-1 is raised and the diagnosis is confirmed with the failure to suppress growth hormone secretion with an oral glucose load. Trans-sphenoidal surgery is first-line management in most instances. If there are ongoing symptoms post-surgery like there are in this case, there are a number of options. Repeat surgery may be indicated if the residual tumour is accessible. However, it is not in this case. Therefore medical management is indicated to control the symptoms. Octreotide is an analogue of the hypothalamic release-inhibiting hormone, somatostatin. It results in significant tumour shrinkage although the mechanism of action by which it exerts this effect is unknown. It is the first-line medical treatment in this situation.

Radiotherapy is incorrect. This treatment is reserved for cases that are refractory to medical management.

Cabergoline is incorrect. This medication was historically used in the medical management of acromegaly. However, it is less effective than octreotide and associated with significant side effects like cardiac fibrosis.

Bromocriptine is incorrect. This medication is less effective than octreotide. It is a dopamine agonist like cabergoline and is associated with a similar side effect profile.

Pegvisomant is incorrect. This is a second line option in the UK for those patients who do not respond to either surgery, radiotherapy or somatostatin analogues. It is an analogue of human GH and a highly selective GH receptor antagonist which blocks the peripheral synthesis of IGF-1.

👍 | 👎 | Discuss (6) | Improve

Next question

Acromegaly: management ★

Trans-sphenoidal surgery is the first-line treatment for acromegaly in the majority of patients.

If a pituitary tumour is inoperable or surgery unsuccessful then medication may be indicated:

- somatostatin analogue
    - directly inhibits the release of growth hormone
    - for example octreotide
    - effective in 50-70% of patients
- pegvisomant
    - GH receptor antagonist - prevents dimerization of the GH receptor

  - once daily s/c administration
  - very effective - decreases IGF-1 levels in 90% of patients to normal
  - doesn't reduce tumour volume therefore surgery still needed if mass effect
- dopamine agonists
  - for example bromocriptine
  - the first effective medical treatment for acromegaly, however now superseded by somatostatin analogues
  - effective only in a minority of patients

External irradiation is sometimes used for older patients or following failed surgical/medical treatment

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

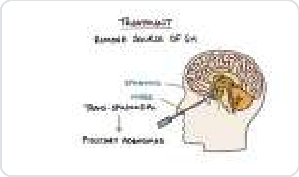
Links

Postgraduate Medical Journal 👍 11 👎 4

Modern treatment of acromegaly

Suggest link Report broken link

Media



Understanding Acromegaly

Zero To Finals - YouTube 👍 7 👎 1

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **67.5%**

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Question 41 of 101

A 38-year-old woman is referred by her GP for management of Graves' disease, diagnosed by the presence of a goitre, suppressed thyroid stimulating hormone, and presence of thyroid antibodies on screening. She has no past medical history of note, drinks 10 units of alcohol per week and smokes 20 cigarettes per day. On examination her blood pressure is 112/88 mmHg, pulse is 89 beats per minute and regular, she has a fine tremor. There is a smooth goitre and marked proptosis.

Which of the following has the greatest negative impact on prognosis of her thyroid eye disease?

- ○ Alcohol consumption ✕
- ○ Cigarette smoking ✕
- ○ DR4 HLA type ✕
- ○ LATS titre ✕
- ○ Use of block replace therapy ✕

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **67.5%**

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41 -

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Which of the following has the greatest negative impact on prognosis of her thyroid eye disease?

Alcohol consumption	3%
Cigarette smoking	85%
DR4 HLA type	5%
LATS titre	2%
Use of block replace therapy	5%

Smoking is the most important modifiable risk factor for the development of thyroid eye disease

Important for me Less important

A systematic review published in 2006 has confirmed the strong link between cigarette smoking and thyroid eye disease. Across 15 studies a strong association between thyroid eye disease in patients with Graves' disease and smoking was established, with an odds ratio of up to 20 for thyroid eye disease in current smokers vs non-smokers who have Graves'.

http://www.ncbi.nlm.nih.gov/pubmed/16980921

Block replace therapy establishes stable control of thyroid function, and is actually associated with reduced incidence of thyroid eye disease because thyroxine is consistently in the normal range. Alcohol consumption within the recommended safe limits may actually reduce the severity of thyroid eye disease in Graves'. Thyroid eye disease is primarily driven by pathogenic T cells, as such it isn't closely related to LATS titre. HLA DR4 is more strongly associated with Type 1 diabetes, rheumatoid arthritis and autoimmune hepatitis than with thyroid disease.

👍 👎 Discuss (1) Improve

Next question

Thyroid eye disease

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology
- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention
- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features
- the patient may be eu-, hypo- or hyperthyroid at the time of presentation

- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management
- smoking cessation
- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery

Complications

Exposure keratopathy
- this is the most common complication of thyroid eye disease
- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy
- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia
- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)

- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):

- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes
- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy 👍 9 👎 8

Management of Graves' Orbitopathy

Suggest link

Report broken link

Score: **68.3%**

1	✓
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Question 42 of 101

An 82-year-old man is reviewed on the ward. He was admitted with recurrent falls four days ago. He has been found to have symptomatic postural hypotension with a drop of 50mmHg on standing in systolic blood pressure.He has a past medical history of hypertension, osteoporosis, radial fracture and COPD. He was taking ramipril and amlodipine, as well as inhalers and calcium supplements. He normally has a good functional baseline and is independent.

Despite stopping all antihypertensives on admission the physiotherapists are unable to mobilise him due to worsening of his symptoms on standing. At rest, he has a blood pressure of 150mmHg systolic. What is the most appropriate plan?

- ○ Fludrocortisone ×
- ○ Hydrocortisone ×
- ○ Discharge bedbound and continue with physiotherapy at home ×
- ○ Discharge bedbound with restarting antihypertensives ×
- ○ Inpatient rehabilitation ×

Submit answer

Reference ranges

Score: **68.3%**

1	✓
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Question 42 of 101 ✗

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Despite stopping all antihypertensives on admission the physiotherapists are unable to mobilise him due to worsening of his symptoms on standing. At rest, he has a blood pressure of 150mmHg systolic. What is the most appropriate plan?

🟩	Fludrocortisone	66%
🟥	Hydrocortisone	22%
	Discharge bedbound and continue with physiotherapy at home	3%
	Discharge bedbound with restarting antihypertensives	2%
	Inpatient rehabilitation	8%

The correct answer is fludrocortisone. Fludrocortisone is a corticosteroid with minimal glucocorticoid activity but very high mineralocorticoid activity which can aid in symptomatic postural hypotension and can significantly reduce symptoms. It may take several days to have an effect but can restore mobility in some patients. Continuing with rehabilitation without addressing his treatable cause would not be optimal, whilst re-introducing his antihypertensives would reduce the risk of stroke and cardiovascular disease but would also reduce his independence significantly.

Discuss (4) Improve

Next question

Corticosteroids

Corticosteroids are amongst the most commonly prescribed therapies in clinical practice. They are used both systemically (oral or intravenous) or locally (skin creams, inhalers, eye drops, intra-articular). They augment and in some cases replace the natural glucocorticoid and mineralocorticoid activity of endogenous steroids.

The relative glucocorticoid and mineralocorticoid activity of commonly used steroids is shown below:

Minimal glucocorticoid activity, very high mineralocorticoid activity,	**Glucocorticoid activity, high mineralocorticoid activity,**	**Predominant glucocorticoid activity, low mineralocorticoid activity**	**Very high glucocorti activity, m mineraloc activity**
Fludrocortisone	Hydrocortisone	Prednisolone	Dexameth Betmetha

Side-effects

The side effects of corticosteroids are numerous and represent the single greatest limitation on their usage. Side-effects are more common with systemic and prolonged therapy.

Glucocorticoid side-effects
- endocrine
    - impaired glucose regulation
    - increased appetite/weight gain
    - hirsutism
    - hyperlipidaemia

- Cushing's syndrome
    - moon face
    - buffalo hump
    - striae
- musculoskeletal
    - osteoporosis
    - proximal myopathy
    - avascular necrosis of the femoral head
- immunosuppression
    - increased susceptibility to severe infection
    - reactivation of tuberculosis
- psychiatric
    - insomnia
    - mania
    - depression
    - psychosis
- gastrointestinal
    - peptic ulceration
    - acute pancreatitis
- ophthalmic
    - glaucoma
    - cataracts
- suppression of growth in children
- intracranial hypertension
- neutrophilia

Mineralocorticoid side-effects

- fluid retention
- hypertension

Selected points on the use of corticosteroids:

- patients on long-term steroids should have their doses doubled during intercurrent illness
- longer-term systemic corticosteroids suppress the natural production of endogenous steroids. They should therefore not be withdrawn abruptly, as this may precipitate an Addisonian crisis
- the BNF suggests gradual withdrawal of systemic corticosteroids if patients have:

  - received more than 40mg prednisolone daily for more than one week
  - received more than 3 weeks of treatment
  - recently received repeated courses

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **66.7%**

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42	✗

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← Question 43 of 101 →

A 68-year-old man was admitted with fever and difficulty passing urine. He had a positive urine dip and was commenced on intravenous co-amoxiclav for a lower urinary tract infection. He also complains of 'burning pain' and ascending numbness from his feet.

His past medical history includes poorly controlled diabetes with retinopathy, hypertension, ischaemic heart disease and hypercholesterolaemia. His medications include metformin, gliclazide, linagliptin, ramipril, aspirin and simvastatin.

Examination of the cardio-respiratory system is normal. He has mild suprapubic tenderness on abdominal palpation. On examination of his feet, you notice decreased sensation and loss of proprioception.

What would be the first-line treatment for the pain the patient is suffering with?

- ○ Codeine ×
- ○ Paracetamol ×
- ○ Tramadol ×
- ○ Ibuprofen ×
- ○ Duloxetine ×

Submit answer

Reference ranges

Score: **66.7%**

1	✓
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43 -

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His past medical history includes poorly controlled diabetes with retinopathy, hypertension, ischaemic heart disease and hypercholesterolaemia. His medications include metformin, gliclazide, linagliptin, ramipril, aspirin and simvastatin.

Examination of the cardio-respiratory system is normal. He has mild suprapubic tenderness on abdominal palpation. On examination of his feet, you notice decreased sensation and loss of proprioception.

What would be the first-line treatment for the pain the patient is suffering with?

Codeine	1%
Paracetamol	4%
Tramadol	4%
Ibuprofen	1%
Duloxetine	91%

First line treatment in diabetic neuropathy is with amitriptyline, duloxetine, gabapentin or pregabalin

Important for me Less important

The patient is suffering from diabetic neuropathy. This is evident from the pain/numbness experienced in the glove-stocking distribution and the abnormal neurology elicited from his feet. First-line treatment for diabetic neuropathic pain includes duloxetine.

Codeine and paracetamol are both analgesics, but would not be of benefit in neuropathic pain.

Tramadol is used as a rescue treatment in acute flares of pain associated with diabetic neuropathy and is not first-line management.

Ibuprofen is also an analgesic that will offer limited benefit in neuropathic pain. The patient is also taking aspirin and dual NSAID use is not advisable due to the risks of gastric ulcers and bleeding.

👍 | 👎 | 💬 Discuss (4) | Improve

Next question

Diabetic neuropathy ★

Peripheral neuropathy

Diabetes typically leads to sensory loss and not motor loss. Sensory loss typically results in a 'glove and stocking' distribution, with the lower legs affected first due to the length of the sensory neurons supplying this area. Painful diabetic neuropathy is also a common problem in clinical practice.

NICE updated it's guidance on the management of neuropathic pain in 2013. Diabetic neuropathy is now managed in the same way as other forms of neuropathic pain:

- first-line treatment: amitriptyline, duloxetine, gabapentin or pregabalin
- if the first-line drug treatment does not work try one of the other 3 drugs
- tramadol may be used as 'rescue therapy' for exacerbations of neuropathic pain

- topical capsaicin may be used for localised neuropathic pain (e.g. post-herpetic neuralgia)
- pain management clinics may be useful in patients with resistant problems

Gastrointestinal autonomic neuropathy

Gastroparesis

- occurs secondary to autonomic neuropathy
- symptoms include erratic blood glucose control, bloating and vomiting
- management options include metoclopramide, domperidone or erythromycin (prokinetic agents)

Chronic diarrhoea

- often occurs at night

Gastro-oesophageal reflux disease

- caused by decreased lower esophageal sphincter (LES) pressure

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 4

2013 Neuropathic pain guidelines

NICE 👍 13 👎 7

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Score: **67.4%**

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← Question 44 of 101 →

A 45-year-old man was commenced on 500mg metformin BD for type 2 diabetes mellitus as he was unable to meet his target HBA1c of 6.5% 6 months ago. He is now presenting for his regular follow up and feels well. He states that he has been compliant with his metformin, maintaining a healthy diet and exercising more regularly. His latest HBA1c is 7.3% and the rest of his investigations are normal. What is the next best step to manage his diabetes?

- ○ Increase his dose of metformin ×
- ○ Add pioglitazone ×
- ○ Make no change to his current treatment ×
- ○ Add a sulfonylurea ×
- ○ Advise to increase frequency of exercise ×

Submit answer

Reference ranges

Score: **67.4%**

1 ✓
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37	✓
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41 ✓
42 ✗
43 ✓
44 -

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Question 44 of 101 ✓

A 45-year-old man was commenced on 500mg metformin BD for type 2 diabetes mellitus as he was unable to meet his target HBA1c of 6.5% 6 months ago. He is now presenting for his regular follow up and feels well. He states that he has been compliant with his metformin, maintaining a healthy diet and exercising more regularly. His latest HBA1c is 7.3% and the rest of his investigations are normal. What is the next best step to manage his diabetes?

Increase his dose of metformin	67%
Add pioglitazone	3%
Make no change to his current treatment	10%
Add a sulfonylurea	20%
Advise to increase frequency of exercise	1%

As per NICE guidelines on the management of type 2 diabetes in adults, at an HBA1c of 7.3% and a relatively low dose of metformin, the first step would be to increase his dose of metformin. His aim should still be an HBA1c of 6.5% on metformin.

Additional medications are only required if his HBA1c is 7.5% or higher, therefore the addition of pioglitazone or a sulfonylurea is incorrect. Whilst regular exercise is important, it is unlikely to be sufficient at this stage.

Click on the link below for more information and the algorithm for blood glucose lowering therapy in adults with type 2 diabetes:

https://www.nice.org.uk/guidance/ng28/chapter/1-Recommendations#drug-treatment-2

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	**HbA1c target**
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

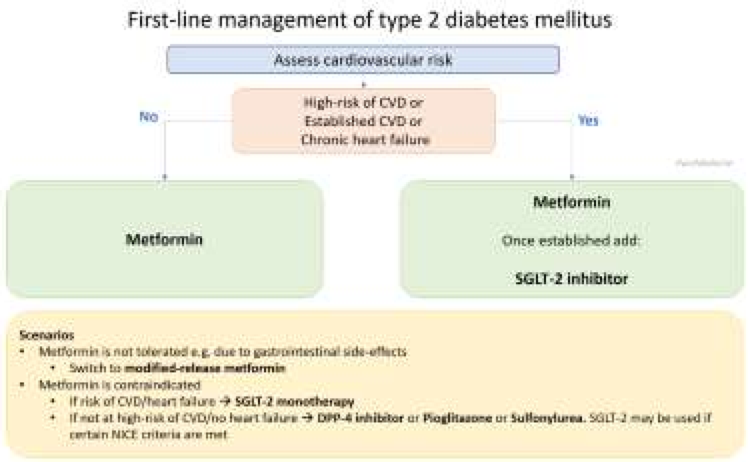
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	**HbA1c target**
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
    - Switch to **modified-release metformin**
- Metformin is contraindicated
    - If risk of CVD/heart failure → **SGLT-2 monotherapy**
    - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

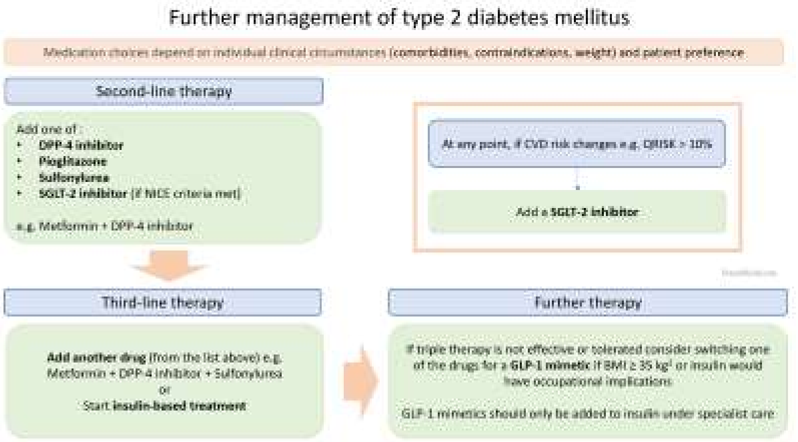
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:

  - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
• DPP-4 inhibitor
• Pioglitazone
• Sulfonylurea
• SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

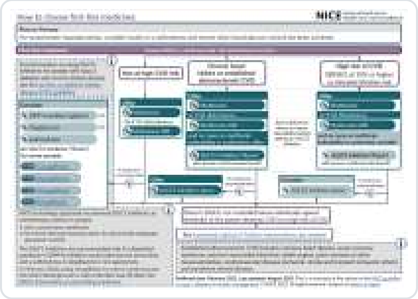
Starting insulin

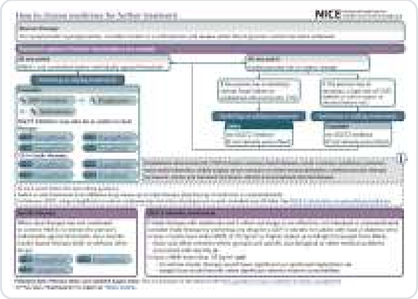
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

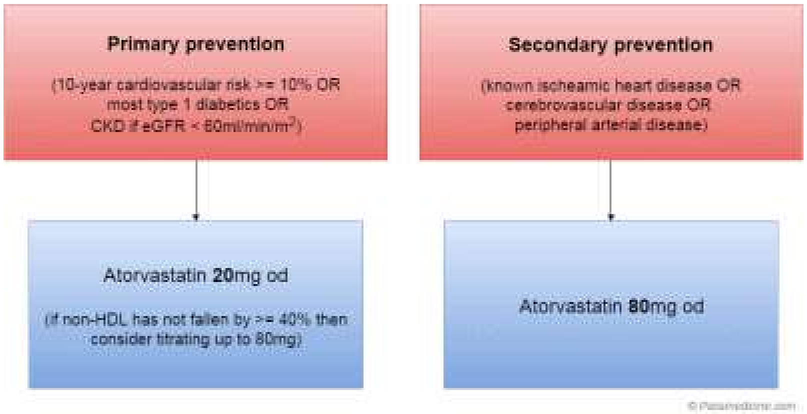
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

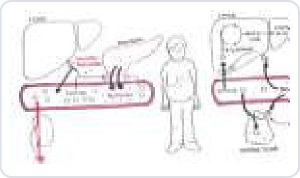
Links

NICE 2014 Lipid modification guidelines	👍 33 👎 40
SIGN 2010 Management of diabetes	👍 36 👎 41
NICE 2022 Type 2 diabetes guidelines	👍 56 👎 48
7 minute medics Basics of type 2 diabetes - podcast	👍 44 👎 35

Suggest link

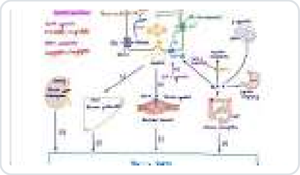
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **68.2%**

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44 ✓

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Question 45 of 101

A patient with type 1 diabetes mellitus is referred urgently from a Dose Adjustment For Normal Eating (DAFNE) course, a course designed to help patients with type 1 diabetes correctly assess carbohydrate contents, to the endocrinology consultant. The nurse in the course was concerned because she found that the patient has had three episodes of hypoglycaemia within the last nine months where he required aid from his wife in order to increase his blood glucose levels. He takes a basal bolus regime of long acting insulin once a night as well as short acting insulin three times a day. He works in a restaurant. He is an ex-smoker who drinks very little alcohol. Apart from adjusting the insulin dose, what is the most appropriate action?

- ○ Advise the patient to inform the DVLA and to not drive ×
- ○ The consultant should inform the DVLA ×
- ○ Advise the patient to start checking blood glucose prior to driving ×
- ○ Advise the patient to always carry a snack when driving ×
- ○ Advise the patient to attend retinal screening prior to driving again ×

Submit answer

Reference ranges

Score: **68.2%**

1	✓
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43	✓
44	✓
45	-

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Question 45 of 101 ✓

A patient with type 1 diabetes mellitus is referred urgently from a Dose Adjustment For Normal Eating (DAFNE) course, a course designed to help patients with type 1 diabetes correctly assess carbohydrate contents, to the endocrinology consultant. The nurse in the course was concerned because she found that the patient has had three episodes of hypoglycaemia within the last nine months where he required aid from his wife in order to increase his blood glucose levels. He takes a basal bolus regime of long acting insulin once a night as well as short acting insulin three times a day. He works in a restaurant. He is an ex-smoker who drinks very little alcohol. Apart from adjusting the insulin dose, what is the most appropriate action?

Advise the patient to inform the DVLA and to not drive	64%
The consultant should inform the DVLA	11%
Advise the patient to start checking blood glucose prior to driving	14%
Advise the patient to always carry a snack when driving	9%
Advise the patient to attend retinal screening prior to driving again	3%

Patient with diabetes who have had two hypoglycaemic episodes requiring help needs to surrender their driving licence

Important for me Less important

This patient has had hypoglycaemic episodes needing help multiple times. If a patient needs help to correct the hypoglycaemic episode then it is termed severe hypoglycaemia and is of concern to the DVLA. If a patient has two or more episodes of severe hypoglycaemia then they need to inform the DVLA and not drive.

Patients who take insulin should have their licence for driving assessed on an annual basis. For this they will be required to submit three months of blood glucose readings. Patients are advised to always check blood glucose before driving and to always have a snack in their vehicle as well, not just if having hypoglycaemic episodes.

When the DVLA is informed, the patient should be advised to inform the DVLA themselves rather than breaking patient confidentiality, but if the patient repeatedly fails to follow this advise then the doctor should inform the DVLA after telling the patient that he or she is doing so.

👍 👎 💬 Discuss (2) Improve

Next question

DVLA: diabetes mellitus

Until recently people with diabetes who used insulin could not hold a HGV licence. The DVLA changed the rules in October 2011. The following standards need to be met (and also apply to patients using other hypoglycaemic inducing drugs such as sulfonylureas):

- there has not been any severe hypoglycaemic event in the previous 12 months
- the driver has full hypoglycaemic awareness
- the driver must show adequate control of the condition by regular blood glucose monitoring*, at least twice daily and at times relevant to driving
- the driver must demonstrate an understanding of the risks of hypoglycaemia
- here are no other debarring complications of diabetes

From a practical point of view patients on insulin who want to apply for a Group 2 (HGV) licence need to complete a VDIAB1I form.

Other specific points for group 1 drivers:

- if on insulin then patient can drive a car as long as they have hypoglycaemic awareness, not more than one episode of hypoglycaemia requiring the assistance of another person within the preceding 12 months and no relevant visual impairment. Drivers are normally contacted by DVLA
- if on tablets or exenatide no need to notify DVLA. If tablets may induce hypoglycaemia (e.g. sulfonylureas) then there must not have been more than one episode of hypoglycaemia requiring the assistance of another person within the preceding 12 months
- if diet controlled alone then no requirement to inform DVLA

*to demonstrate adequate control, the Secretary of State's Honorary Medical Advisory Panel on Diabetes Mellitus has recommended that applicants will need to have used blood glucose meters with a memory function to measure and record blood glucose levels for at least 3 months prior to submitting their application

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DVLA 👍 7 👎 9

Fitness to drive guidelines

Gov.uk 👍 12 👎 4

Diabetes and driving

Suggest link Report broken link

Score: **68.9%**

1	✓
2	✓
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45	✓

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Question 46 of 101

You are asked to review a 62 year-old Caucasian man who is an inpatient on the medical admissions unit. He is currently being treated for a left lower lobe community acquired pneumonia. You note consumed alcohol excessively prior to admission but has been abstinent for the last four days.

During this admission it has been noted that serial bloods glucose measurements have been elevated and subsequently a new diagnosis type two diabetes has been made. The admission consultant noted Cushingoid featured and requested an overnight low dose dexamethasone suppression test. The results are as follows:

8am Cortisol after 1mg dexamethasone at 11pm the previous day	438 nmol/L
Reference range for serum cortisol	170-540 nmol/L

What is most appropriate next step in the investigation of this gentleman?

- ○ Serum ACTH measurement ×
- ○ Midnight serum cortisol ×
- ○ Inferior petrosal sinus sampling post CRH administration ×
- ○ High dose dexamethasone suppression test ×
- ○ MRI pituitary ×

Submit answer

Reference ranges

Score: **68.9%**

1	✓
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7	✗
8	✓
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11	✗
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42	✗
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46	-

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8am Cortisol after 1mg dexamethasone at 11pm the previous day	438 nmol/L
Reference range for serum cortisol	170-540 nmol/L

What is most appropriate next step in the investigation of this gentleman?

	Serum ACTH measurement	24%
🟩	Midnight serum cortisol	8%
	Inferior petrosal sinus sampling post CRH administration	3%
🟥	High dose dexamethasone suppression test	63%
	MRI pituitary	2%

Pseudo-Cushing's syndrome is common in those with excessive alcohol consumption. The physical signs resemble true Cushing's syndrome however the aetiology is idiopathic rather than dysfunction of the hypothalamic-pituitary axis. It is therefore important to exclude pseudo-Cushing's prior to further investigation.

The hallmark of true Cushing's syndrome is lack of diurnal variation in serum cortisol. However in pseudo-Cushing's diurnal variation is normally maintained. Those with pseudo-Cushing's will have elevated 24 hour urinary cortisol and will also fail to suppress serum cortisol with a low dose dexamethasone suppression test. The correct approach in this case is therefore to measure a midnight cortisol prior to proceeding to further investigation.

Papanicolaou DA, Yanovski JA, Cutler GB Jr. A single midnight serum cortisol measurement distinguishes Cushing's syndrome from pseudo-Cushing states. J Clin Endocrinol Metab. 1998 Apr. 83(4):1163-7.

👍 👎 Discuss (10) Improve

Next question

Cushing's syndrome: investigations

This section will consider:

- the general lab findings consistent with Cushing's syndrome
- tests to confirm whether a patient indeed has Cushing's syndrome
- tests to find the underlying cause of Cushing's syndrome

It is useful to bear in mind the possible causes:

- iatrogenic: corticosteroid therapy
- ACTH-dependent causes
    - Cushing's disease (a pituitary adenoma → ACTH secretion)
    - ectopic ACTH secretion secondary to a malignancy
- ACTH-independent causes
    - adrenal adenoma

General findings consistent with Cushing's syndrome

A hypokalaemic metabolic alkalosis may be seen, along with impaired glucose tolerance.

Ectopic ACTH secretion (e.g. secondary to small cell lung cancer) is characteristically associated with very low potassium levels.

Tests to confirm Cushing's syndrome

The three most commonly used tests are:

- overnight (low-dose) dexamethasone suppression test
    - this is the most sensitive test and is now used first-line to test for Cushing's syndrome
    - patients with Cushing's syndrome do not have their morning cortisol spike suppressed
- 24 hr urinary free cortisol
    - two measurements are required
- bedtime salivary cortisol
    - two measurements are required

Localisation tests

The first-line localisation is 9am and midnight plasma ACTH (and cortisol) levels. If ACTH is suppressed then a non-ACTH dependent cause is likely such as an adrenal adenoma

High-dose dexamethasone suppression test

The high-dose dexamethasone suppression test may be used to localise the pathology resulting in Cushing's syndrome. This test may be interpreted as follows:

Cortisol	ACTH	Interpretation
Not suppressed	Suppressed	Cushing's syndrome due to other causes (e.g. adrenal adenomas)
Suppressed	Suppressed	Cushing's disease (i.e. pituitary adenoma → ACTH secretion)

Cortisol	ACTH	Interpretation
Not suppressed	Not suppressed	Ectopic ACTH syndrome

Other tests

CRH stimulation
- if pituitary source then cortisol rises
- if ectopic/adrenal then no change in cortisol

Petrosal sinus sampling of ACTH may be needed to differentiate between pituitary and ectopic ACTH secretion.

An insulin stress test is used to differentiate between true Cushing's and pseudo-Cushing's.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypothalamus
Anterior Pituitary
Adrenal Gland
Kidney
Dexamethasone 1mg
(Cushing's Syndrome)
ZERO TO FINALS

[Understanding The Dexamethasone Suppression Test]

Zero to Finals - YouTube 👍 31 👎 3

CUSHING SYNDROME

[Cushing syndrome]

Osmosis - YouTube 👍 7 👎 1

CUSHING'S SYNDROME

[Understanding Cushing's Syndrome]

Zero To Finals - YouTube 👍 1 👎 1

Report broken media

Score: **67.4%**

1 ✓
2 ✓
3 ✗
4 ✓
5 ✗
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7 ✗
8 ✓

9	✓
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44 ✓

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46 ✗

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A 34-year-old woman calls the endocrinology nurse for advice. She has been vomiting for the last 24 hours and has been unable to take her regular medications. She has a past medical history of Addison's disease. She normally takes oral hydrocortisone and fludrocortisone. Apart from vomiting and reduced oral intake she denies any symptoms. Specifically, she has not been dizzy on standing, had any blackouts or any diarrhoea. She has also been having a normal temperature. She has IM hydrocortisone at home.

What is the most appropriate advice to give in regards to her hydrocortisone?

- ○ Advise her to come to the emergency department ×
- ○ Advise her to take the IM hydrocortisone at come to the emergency department for review ×
- ○ Advise her to not take any hydrocortisone until the vomiting settles ×
- ○ Adviser her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms ×
- ○ Advise her to double the dose of oral hydrocortisone ×

Submit answer

Reference ranges

Score: **67.4%**

1	✓
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What is the most appropriate advice to give in regards to her hydrocortisone?

	Advise her to come to the emergency department	4%
🟩	Advise her to take the IM hydrocortisone at come to the emergency department for review	75%
	Advise her to not take any hydrocortisone until the vomiting settles	1%
	Adviser her to take IM hydrocortisone only if starting to feel faint or experiencing postural symptoms	5%
🟥	Advise her to double the dose of oral hydrocortisone	14%

A person with Addisons' who vomits should take IM hydrocortisone until vomiting stops

Important for me Less important

This is a patient with Addison's disease who is unable to take her regular oral hydrocortisone and is therefore at risk of developing an Addisonian crisis. If unable to take the normal oral hydrocortisone then the patient should be advised to take IM hydrocortisone to avoid this. This is why all patients with Addison's disease should have IM hydrocortisone for these situations.

Given that she has been vomiting for the past 24 hours it is appropriate for her to attend the emergency department for assessment and possible IV fluids, IV hydrocortisone.

👍 👎 Discuss Improve

Next question

Addison's disease: management ★

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:
- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:
- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards
- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 19 👎 16

Addison's disease

Royal College of Physicians 👍 16 👎 8

2017 Adrenal insufficiency â€“ recognition and management

Suggest link

Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube

👍 1 👎 0

[Primary adrenal insufficiency (Addison's disease)](#)

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **66%**

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41	✓
42	✗
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46	✗
47	✗

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A 64-year-old woman attends the emergency department with heart palpitations. She has a past medical history of type 2 diabetes mellitus. Observations are as follows: heart rate 92 beats per minute, and blood pressure 145/85 mmHg,

Blood results are as follows:

Hb	116 g/L	Male: (135-180) Female: (115 - 160)
Platelets	156 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	6.5 mmol/L	(3.5 - 5.0)
Cl^-	125 mmol/L	(96 - 106)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	115 µmol/L	(55 - 120)
Bicarbonate	12 mmol/L	(23 - 29)
Glucose	34.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

- ○ Addison's disease ×
- ○ Diabetic ketoacidosis (DKA) ×
- ○ Type 1 renal tubular acidosis ×
- ○ Type 2 renal tubular acidosis ×
- ○ Type 4 renal tubular acidosis ×

Submit answer

Reference ranges ⌄

Score: **66%**

1	✓
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47	✗
48	-

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Glucose	34.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

○ Addison's disease ×

○ Diabetic ketoacidosis (DKA) ×

○ Type 1 renal tubular acidosis ×

○ Type 2 renal tubular acidosis ×

○ Type 4 renal tubular acidosis ×

Submit answer

Reference ranges ⌵

Score: **66%**

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43	✓
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48	-

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Back to top

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Bicarbonate	12 mmol/L	(23 - 29)
Glucose	34.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

Addison's disease	3%
Diabetic ketoacidosis (DKA)	20%
Type 1 renal tubular acidosis	4%
Type 2 renal tubular acidosis	3%
[✓ Type 4 renal tubular acidosis]	70%

Renal tubular acidosis (type 4) causes hyperkalaemia

Important for me Less important

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 6.5) - (125 + 12) = 5.5.

Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include ('ABCD'):

- Addison's
- Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
- Chloride excess
- Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:

- Lactate
- Toxins (e.g. methanol, paracetamol, propylene glycol)
- Ketones
- Renal failure

Type 4 renal tubular acidosis is correct. The presence of NAGMA, hyperkalemia, and a history of diabetes favours a diagnosis of type 4 renal tubular acidosis.

Type 1 renal tubular acidosis is incorrect. Although this condition also presents with NAGMA, it is associated with hypokalemia.

Addison's disease is incorrect. Addison's can result in NAGMA, with hyperkalemia as a consequence of hypoaldosteronism. However, the presence of hyperglycaemia and hypertension makes this a less likely diagnosis. One would expect hypoglycaemia and hypotension in

Addison's.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This condition is associated with NAGMA and an hypokalemia.

👍 | 👎 | Discuss (4) | Improve

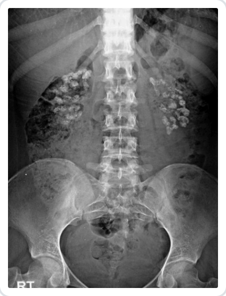
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia

- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

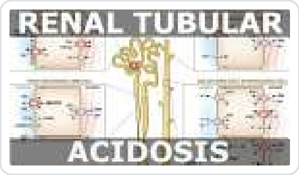
Extended textbook

Media

RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

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Question 49 of 101

A 51-year-old woman is reviewed in clinic. Two months ago she underwent an operation to remove a medullary thyroid cancer after presenting with diarrhoea and a neck lump. Genetic testing showed she has a mutation of the RET oncogene. The patient reports being well and there are no signs of local recurrence on examination.

What is the most appropriate test to monitor for recurrence?

- ○ Thyroglobulin ✕
- ○ Thyroid transcription factor-1 ✕
- ○ Chromogranin ✕
- ○ Calcitonin ✕
- ○ S100 protein ✕

Submit answer

Reference ranges

Score: **66.7%**

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49 -

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Question 49 of 101 ✓

A 51-year-old woman is reviewed in clinic. Two months ago she underwent an operation to remove a medullary thyroid cancer after presenting with diarrhoea and a neck lump. Genetic testing showed she has a mutation of the RET oncogene. The patient reports being well and there are no signs of local recurrence on examination.

What is the most appropriate test to monitor for recurrence?

Thyroglobulin	23%
Thyroid transcription factor-1	1%
Chromogranin	1%
Calcitonin	74%
S100 protein	1%

Medullary thyroid cancer - calcitonin is used for screening, prognosis and monitoring

Important for me Less important

S100 protein is used in patients with melanoma. Thyroglobulin is used in other types of thyroid malignancy but not medullary thyroid cancer.

Discuss (2) Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	Percentage	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare

Type	**Notes**
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

Suggest link

Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0

Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **67.3%**

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← Question 50 of 101 →

An 18-year-old man comes to the endocrinology clinic for review. He has been transferred from the paediatric clinic with a diagnosis of congenital hypoparathyroidism and is treated with vitamin D and calcium supplementation. He has had one episode of symptomatic renal stones over the past 3 years and his creatinine is elevated at 125 micromol/l.

Which of the following is the most appropriate target with respect to serum calcium?

- ○ 1.85 mmol/l ×
- ○ 2.10 mmol/l ×
- ○ 2.25 mmol/l ×
- ○ 2.60 mmol/l ×
- ○ 2.85 mmol/l ×

Submit answer

Reference ranges

Score: **67.3%**

1 ✓
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An 18-year-old man comes to the endocrinology clinic for review. He has been transferred from the paediatric clinic with a diagnosis of congenital hypoparathyroidism and is treated with vitamin D and calcium supplementation. He has had one episode of symptomatic renal stones over the past 3 years and his creatinine is elevated at 125 micromol/l.

Which of the following is the most appropriate target with respect to serum calcium?

	1.85 mmol/l	7%
🟩	2.10 mmol/l	41%
🟥	2.25 mmol/l	40%
	2.60 mmol/l	10%
	2.85 mmol/l	2%

This 18-year-old man is at risk of symptomatic hypocalcaemia because of congenital hypoparathyroidism, although elevating his calcium too much with vitamin D supplementation runs the risk of symptomatic renal stones. As such guidelines recommend aiming towards a calcium just below the lower end of the normal range and 2.10 is an appropriate target for serum calcium.

1.85 mmol/l is considered too low and puts the patient at risk of muscle weakness, paresthesias, tetany and cardiac arrhythmia. Maintenance of calcium either at the upper end of the normal range, (2.60 mmol/l), or 2.85 mmol/l (above the normal range), is associated with symptomatic renal stones and progressive deterioration in renal function.

The European Endocrine Society Guidelines support his calcium target: (page G2) http://www.eje-online.org/content/173/2/G1.full.pdf

👍 👎 Discuss (4) Improve

Next question

Hypoparathyroidism

Primary hypoparathyroidism
- decrease PTH secretion
- e.g. secondary to thyroid surgery*
- low calcium, high phosphate
- treated with alfacalcidol

The main symptoms of hypoparathyroidism are secondary to hypocalcaemia:
- tetany: muscle twitching, cramping and spasm
- perioral paraesthesia
- Trousseau's sign: carpal spasm if the brachial artery occluded by inflating the blood pressure cuff and maintaining pressure above systolic
- Chvostek's sign: tapping over parotid causes facial muscles to twitch
- if chronic: depression, cataracts
- ECG: prolonged QT interval

Pseudohypoparathyroidism
- target cells being insensitive to PTH
- due to abnormality in a G protein
- associated with low IQ, short stature, shortened 4th and 5th metacarpals
- low calcium, high phosphate, high PTH
- diagnosis is made by measuring urinary cAMP and phosphate levels following an infusion of PTH. In hypoparathyroidism this will cause

an increase in both cAMP and phosphate levels. In pseudohypoparathyroidism type I neither cAMP nor phosphate levels are increased whilst in pseudohypoparathyroidism type II only cAMP rises.

Pseudopseudohypoparathyroidism

- similar phenotype to pseudohypoparathyroidism but normal biochemistry

*this may seem an oxymoron, but most medical textbooks classify hypoparathyroidism which is secondary to surgery as being 'primary hypoparathyroidism'

AI + Q 123

Next question

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **66%**

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Question 51 of 101

A 43-year-old female presents with neck discomfort worsening over the past 2 months. She has no other past medical or family history. Examination reveals a firm neck lump moving with swallowing but not with tongue protrusion. Subsequent ultrasound of her neck with fine needle aspirate reveals a 2.5cm papillary thyroid carcinoma. A CT neck reveals one single lymph node in her left anterior cervical chain. What is the optimum treatment?

- ○ Thyroidectomy and neck dissection with postoperative radioiodine ablation ✕
- ○ Thyroidectomy and neck dissection without postoperative radioiodine ablation ✕
- ○ Lobectomy and neck dissection with postoperative radioiodine ablation ✕
- ○ Lobectomy and neck dissection without postoperative radioiodine ablation ✕
- ○ Monitor annually ✕

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **66%**

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Question 51 of 101 ✓

A 43-year-old female presents with neck discomfort worsening over the past 2 months. She has no other past medical or family history. Examination reveals a firm neck lump moving with swallowing but not with tongue protrusion. Subsequent ultrasound of her neck with fine needle aspirate reveals a 2.5cm papillary thyroid carcinoma. A CT neck reveals one single lymph node in her left anterior cervical chain. What is the optimum treatment?

Thyroidectomy and neck dissection with postoperative radioiodine ablation	80%
Thyroidectomy and neck dissection without postoperative radioiodine ablation	5%
Lobectomy and neck dissection with postoperative radioiodine ablation	12%
Lobectomy and neck dissection without postoperative radioiodine ablation	2%
Monitor annually	1%

Diagnosis of thyroid tumours are frequently made after the patient has self-palpated a neck lump or after an incidental finding following unrelated neck imaging. Most prevalent in young females, diagnosis is clinched on fine needle aspiration. The key considerations for treatment are the size of the thyroid mass and the presence of lymph node involvement: any lump greater than 1cm in size or has any signs of metastatic spread should undergo thyroidectomy instead of a lobectomy. The postoperative use of concurrent radioiodine ablation enhances survival benefits in patients at high risk of disease recurrence and is recommended by both the American Thyroid association and a European consensus group. Patients with thyroid lumps greater than 4cm in diameter regardless of extrathyroid disease; those with lumps between 1 and 4cm in diameter and extra-thyroid disease; and all with high-risk histology such as aggressive histological subtypes benefit from radioiodine ablation. In the case of this patient with a 2.5cm mass and lymph node involvement, thyroidectomy, lymph node clearance with neck dissection and postoperative radioiodine ablation is appropriate. Annual monitoring is thus an unsafe option.

👍 👎 Discuss (7) Improve

Next question

Thyroid cancer

Features of hyperthyroidism or hypothyroidism are not commonly seen in patients with thyroid malignancies as they rarely secrete thyroid hormones

Main points

Type	**Percentage**	
Papillary	70%	Often young females - excellent prognosis
Follicular	20%	
Medullary	5%	Cancer of parafollicular (C) cells, secrete calcitonin, part of MEN-2
Anaplastic	1%	Not responsive to treatment, can cause pressure symptoms
Lymphoma	Rare	Associated with Hashimoto's thyroiditis

Management of papillary and follicular cancer

- total thyroidectomy
- followed by radioiodine (I-131) to kill residual cells
- yearly thyroglobulin levels to detect early recurrent disease

Further information

Type	Notes
Papillary carcinoma	• Usually contain a mixture of papillary and colloidal filled follicles • Histologically tumour has papillary projections and pale empty nuclei • Seldom encapsulated • Lymph node metastasis predominate • Haematogenous metastasis rare
Follicular adenoma	• Usually present as a solitary thyroid nodule • Malignancy can only be excluded on formal histological assessment
Follicular carcinoma	• May appear macroscopically encapsulated, microscopically capsular invasion is seen. Without this finding the lesion is a follicular adenoma. • Vascular invasion predominates • Multifocal disease raree
Medullary carcinoma	• C cells derived from neural crest and not thyroid tissue • Serum calcitonin levels often raised • Familial genetic disease accounts for up to 20% cases • Both lymphatic and haematogenous metastasis are recognised, nodal disease is associated with a very poor prognosis.
Anaplastic carcinoma	• Most common in elderly females • Local invasion is a common feature • Treatment is by resection where possible, palliation may be achieved through

Type	Notes
	isthmusectomy and radiotherapy. Chemotherapy is ineffective.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thyroid Association 👍 7 👎 6

2014 Guidelines for the Management of Thyroid Cancer

Suggest link

Report broken link

Media

THYROID CANCER

Thyroid cancer

Osmosis - YouTube 👍 5 👎 0

Thyroid Nodule

Armando Hasudungan - YouTube 👍 9 👎 3

Report broken media

Score: **66.7%**

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Question 52 of 101

A 62-year-old man comes to the Emergency department with nausea and vomiting which has steadily worsened over the past 2-3 weeks. He had Type 2 diabetes for the past 7 years and is currently treated with metformin, sitagliptin and empagliflozin. He tells you he has lost some 5kg in weight over the past month. On examination his blood pressure is 110/65 mmHg, his pulse is 85 beats per minute and regular. Emergency blood testing reveal elevated ketones and a glucose of 12.2 mmol/l.

Which of the following is the most appropriate way to manage his glucose control?

- ○ Add liraglutide ×
- ○ Add long-acting insulin ×
- ○ Change the empagliflozin for liraglutide ×
- ○ Change the empagliflozin for long-acting insulin ×
- ○ Stop the metformin ×

Submit answer

Reference ranges

Score: **66.7%**

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← Question 52 of 101 ✕ ⚐ →

A 62-year-old man comes to the Emergency department with nausea and vomiting which has steadily worsened over the past 2-3 weeks. He had Type 2 diabetes for the past 7 years and is currently treated with metformin, sitagliptin and empagliflozin. He tells you he has lost some 5kg in weight over the past month. On examination his blood pressure is 110/65 mmHg, his pulse is 85 beats per minute and regular. Emergency blood testing reveal elevated ketones and a glucose of 12.2 mmol/l.

Which of the following is the most appropriate way to manage his glucose control?

	Add liraglutide	3%
	Add long-acting insulin	18%
🟥	Change the empagliflozin for liraglutide	25%
🟩	Change the empagliflozin for long-acting insulin	42%
	Stop the metformin	11%

Given the duration of Type 2 diabetes and the fact that patient has lost weight in the past month, the possibility that he is insulinopenic is raised. In this situation, calorie loss and metabolic disturbance can be exacerbated by the use of SGLT-2 inhibitors and patients may present as here, with euglycaemic ketoacidosis. The SGLT-2 inhibitor should be withdrawn, and given he is insulinopaenic, long-acting insulin added.

In this situation the empagliflozin must be withdrawn, therefore options including adding liraglutide and long-acting insulin are incorrect. GLP-1 agonists such as liraglutide work less well in patients who are relatively insulinopenic, so liraglutide is incorrect. Stopping the metformin won't remove the cause of ketosis, the empagliflozin.

http://care.diabetesjournals.org/content/38/9/1638

👍 👎 Discuss (12) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

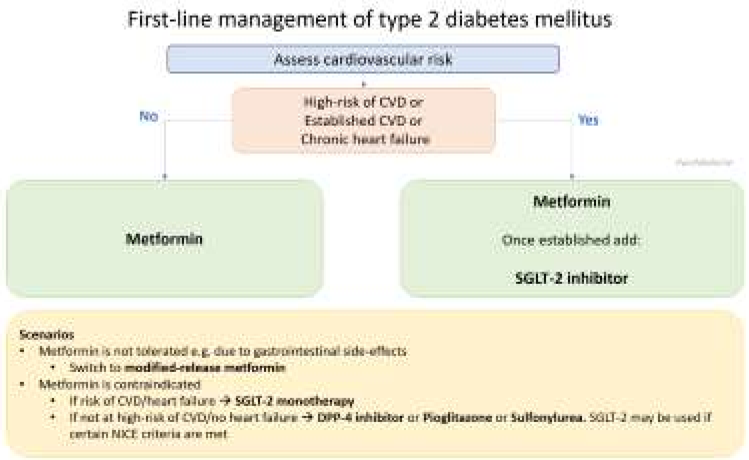
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
  - Switch to **modified-release metformin**
- Metformin is contraindicated
  - If risk of CVD/heart failure → **SGLT-2 monotherapy**
  - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

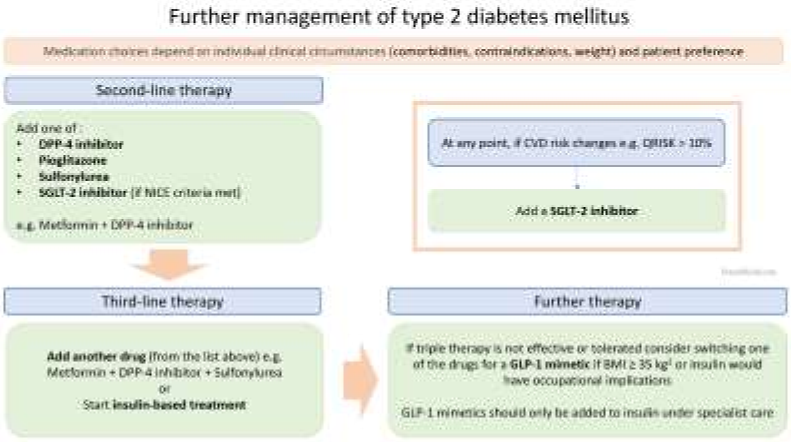
SGLT-2 inhibitors

- should also be given in addition to metformin if any of the following apply:
  - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
  - the patient has established CVD
  - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:

- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

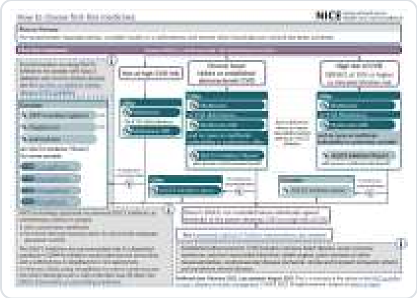
Starting insulin

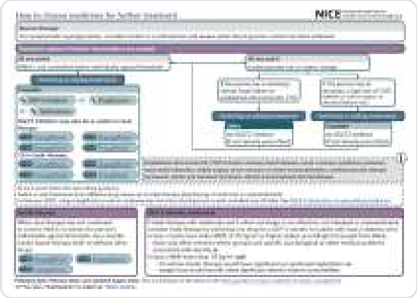
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)

- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
  - an ARB is preferred if the patient has a black African or African-Caribbean family origin

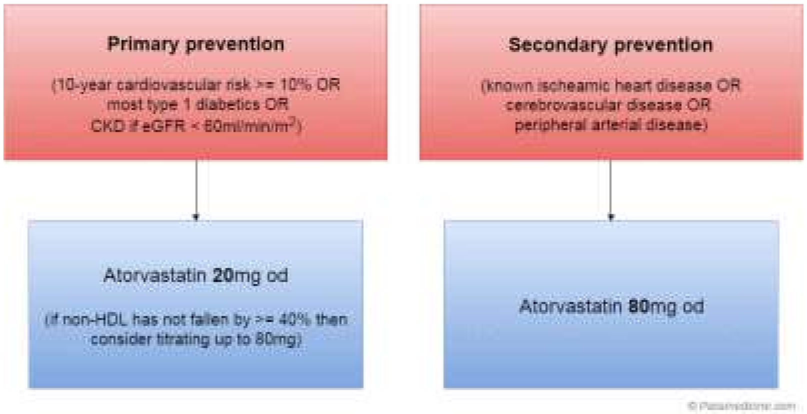
	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR
most type 1 diabetics OR
CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR
cerebrovascular disease OR
peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then
consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

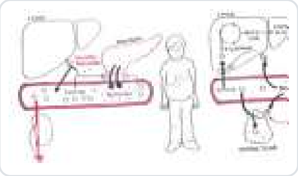
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

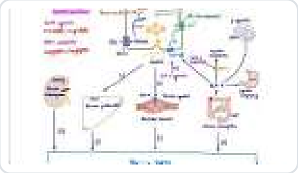
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **65.4%**

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Question 53 of 101

A 64-year-old man presents to his general practitioner with polyuria and polydipsia. He has a past medical history of ischaemic heart disease and mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS syndrome). He takes aspirin, atenolol and atorvastatin.

The examination is unremarkable.

Blood tests:

Hb	133 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.6 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	22 mmol/L	(2.0 - 7.0)
Creatinine	210 µmol/L	(55 - 120)
eGFR	31 mL/minute	(> 90)
CRP	4 mg/L	(< 5)
HbA1c	56 mmol/mol	(<48)

What is the most appropriate choice of management?

○ Empagliflozin ×

○ Gliclazide ×

○ Metformin ×

○ Pioglitazone ×

○ Sitagliptin ×

Submit answer

Reference ranges

Score: **65.4%**

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A 64-year-old man presents to his general practitioner with polyuria and polydipsia. He has a past medical history of ischaemic heart disease and mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS syndrome). He takes aspirin, atenolol and atorvastatin.

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eGFR	31 mL/minute	(> 90)
CRP	4 mg/L	(< 5)
HbA1c	56 mmol/mol	(<48)

What is the most appropriate choice of management?

🟩	Empagliflozin	60%
	Gliclazide	14%
🟥	Metformin	10%
	Pioglitazone	1%

Sitagliptin 15%

T2DM initial therapy: if metformin is contraindicated + patient has a risk of CVD, established CVD or chronic heart failure → SGLT-2 monotherapy

Important for me Less important

Empagliflozin is correct. The patient has developed type 2 diabetes. Metformin is contraindicated in lactic acidosis, so it would be best to avoid it in a patient with MELAS syndrome. As the patient has established CVD, empagliflozin is the most suitable choice of initial anti-diabetic medication. It can be started if the eGFR is > 30ml/minute in those with established CVD.

Gliclazide is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

Metformin is incorrect. Metformin is contraindicated in lactic acidosis, so it would be best to avoid it in a patient with MELAS syndrome.

Pioglitazone is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

Sitagliptin is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

👍 👎 Discuss (10) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

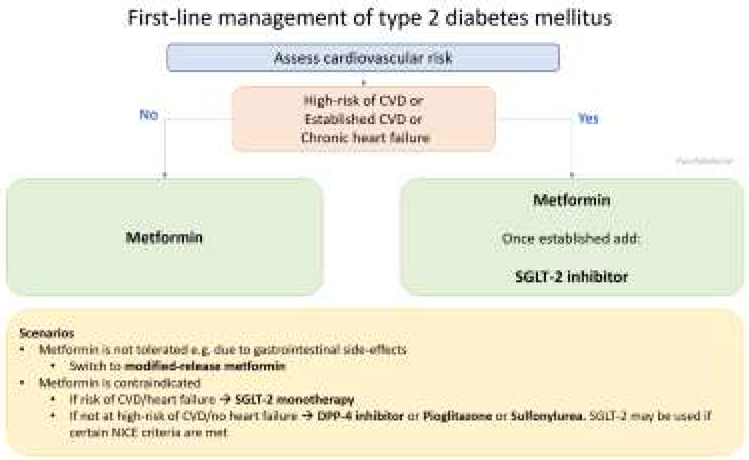
Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

Practical examples
- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

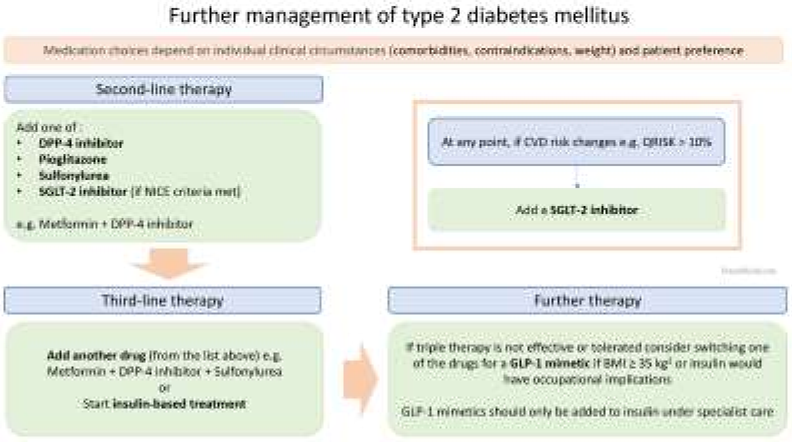
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:

  - DPP-4 inhibitor or pioglitazone or a sulfonylurea
  - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus

Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference

Second-line therapy

Add one of :
- **DPP-4 inhibitor**
- **Pioglitazone**
- **Sulfonylurea**
- **SGLT-2 inhibitor** (if NICE criteria met)

e.g. Metformin + DPP-4 inhibitor

At any point, if CVD risk changes e.g. QRISK > 10%

Add a **SGLT-2 inhibitor**

Third-line therapy

Add another drug (from the list above) e.g.
Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start **insulin-based treatment**

Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a **GLP-1 mimetic** if BMI ≥ 35 kg^2 or insulin would have occupational implications

GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:
- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea

- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
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Further therapy

If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
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- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples
- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
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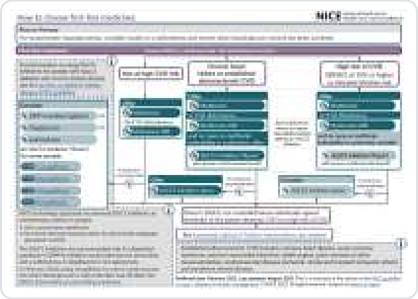
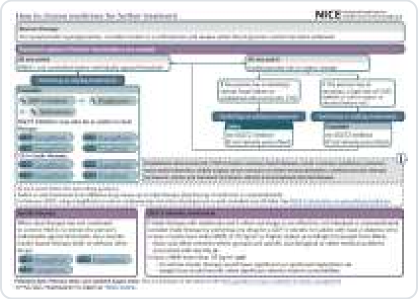




Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line

    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

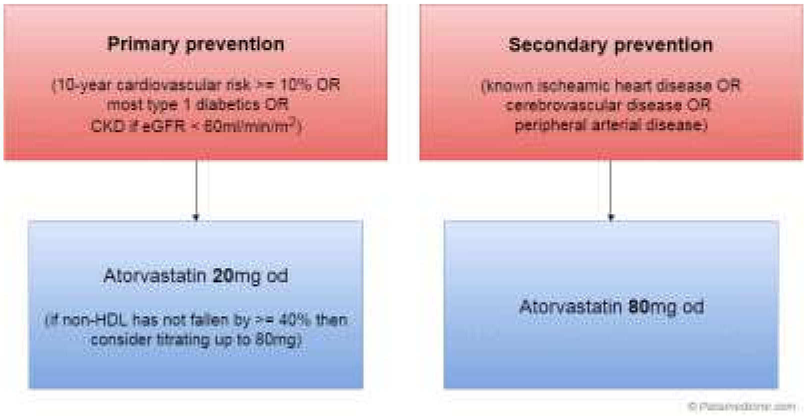
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on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Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
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Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

2022 Type 2 diabetes guidelines

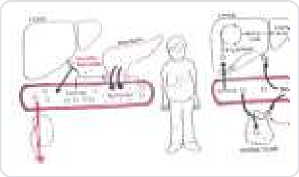
7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

Suggest link

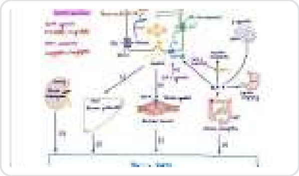
Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

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Score: **64.2%**

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← Question 54 of 101 →

A 38-year-old woman is referred to the outpatient department by her GP with pain in her calves when walking 50 meters. She reports no other symptoms and has no other past medical history other than migraine. She is on no regular medication and her family history includes her mother having diabetes and her father dying of a heart attack aged 46. She currently smokes 35 cigarettes per day and drinks a glass of wine every evening. Her occupation is as a financial advisor.

Examination reveals tendon xanthomas affecting the extensor tendons of his fingers. On examining her face, it is noticed she has xanthelasma around both eyes and corneal arcs.

Which of the following is the most likely diagnosis?

- ○ Tangier disease ✕
- ○ Homozygous familial hypercholesterolaemia ✕
- ○ Heterozygous familial hypercholesterolaemia ✕
- ○ Familial hypertriglyceridaemia ✕
- ○ Apo CII deficiency ✕

Submit answer

Reference ranges

Score: **64.2%**

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Examination reveals tendon xanthomas affecting the extensor tendons of his fingers. On examining her face, it is noticed she has xanthelasma around both eyes and corneal arcs.

Which of the following is the most likely diagnosis?

	Tangier disease	1%
	Homozygous familial hypercholesterolaemia	35%
🟩	Heterozygous familial hypercholesterolaemia	42%
	Familial hypertriglyceridaemia	20%
	Apo CII deficiency	1%

This patient's symptoms suggest intermittent claudication and combined with the signs of hypercholesterolaemia especially the tendon xanthomas would suggest one of the familial hypercholesterolaemias. Patients with homozygous familial hypercholesterolaemia, present with early cardiovascular disease, sometimes as early as the second decade of life, whereas, patients with heterozygous familial hypercholesterolaemia rarely present before the age 30. Thus, this case is more suggestive of heterozygous familial hypercholesterolaemia.

In heterozygous familial hypercholesterolaemia, total cholesterol would typically be above 7.9 mmol/l, with normal triglyceride levels. In homozygotes it is typically above 15 mmol/l.

👍 👎 Discuss (6) Improve

Next question

Familial hypercholesterolaemia ★

Familial hypercholesterolaemia (FH) is an autosomal dominant condition that is thought to affect around 1 in 500 people. It results in high levels of LDL-cholesterol which, if untreated, may cause early cardiovascular disease (CVD). FH is caused by mutations in the gene which encodes the LDL-receptor protein.

Case finding:

- NICE suggest that we should suspect FH as a possible diagnosis in adults with:
    - a total cholesterol level greater than 7.5 mmol/l and/or
    - a personal or family history of premature coronary heart disease (an event before 60 years in an index individual or first-degree relative)
- children of affected parents:
    - if one parent is affected by familial hypercholesterolaemia, arrange testing in children by age 10
    - if both parents are affected by familial hypercholesterolaemia, arrange testing in children by age 5

Clinical diagnosis is now based on the **Simon Broome criteria**:

- in adults total cholesterol (TC) > 7.5 mmol/l and LDL-C > 4.9 mmol/l or children TC > 6.7 mmol/l and LDL-C > 4.0 mmol/l, plus:
- for definite FH: tendon xanthoma in patients or 1st or 2nd degree relatives or DNA-based evidence of FH
- for possible FH: family history of myocardial infarction below age 50 years in 2nd degree relative, below age 60 in 1st degree relative, or a family history of raised cholesterol levels

Management

- the use of CVD risk estimation using standard tables is not appropriate in FH as they do not accurately reflect the risk of CVD
- referral to a specialist lipid clinic is usually required
- high-dose statins are usually used first-line
- first-degree relatives have a 50% chance of having the disorder and should therefore be offered screening. This includes children who should be screened by the age of 10 years if there is one affected parent
- statins should be discontinued in women 3 months before conception due to the risk of congenital defects

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The FH Foundation 👍 5 👎 4

High cholesterol during preganancy

The FH Foundation 👍 7 👎 5

Simon Broom Criteria

Public Library of Science (PLoS) 👍 5 👎 3

Cascade Screening for Familial Hypercholesterolemia (FH)

NICE 👍 4 👎 2

2008 Familial hypercholesterolaemia guidelines

Suggest link Report broken link

Media

FAMILIAL
HYPERCHOLESTEROLEMIA

Familial hypercholesterolaemia

Osmosis - YouTube 👍 10 👎 1

Report broken media

Score: **64.8%**

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Question 55 of 101

A 29-year-old woman is referred by her GP to the outpatient department with increasing symptoms of heat intolerance, diarrhoea and anxiety over the past couple of weeks. The patient is 34 weeks pregnant with her first baby and has a past medical history of hyperthyroidism, currently being treated with 10mg carbimazole. She has no other past medical history of note and her mother also had hyperthyroidism. She does not smoke or drink alcohol and does not take any recreational drugs.

On examination, her pulse is 98 beats per minute, blood pressure is 124/82 mmHg and her respiratory rate is 14/min. Her oxygen saturation is 98% and temperature is 37.5ºC.

Blood tests are performed and reveal:

Thyroid stimulating hormone (TSH)	0.04 mu/l
Free thyroxine (T4)	21 pmol/l
Total thyroxine (T4)	152 nmol/l

What is the most appropriate management?

- ○ Refer patient for immediate caesarean section ×
- ○ Increase carbimazole dose to 20mg once daily ×
- ○ Commence radioiodine treatment ×
- ○ Switch carbimazole to propylthiouracil ×
- ○ Refer for a thyroidectomy ×

Submit answer

Reference ranges

Score: **64.8%**

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Blood tests are performed and reveal:

Thyroid stimulating hormone (TSH)	0.04 mu/l
Free thyroxine (T4)	21 pmol/l
Total thyroxine (T4)	152 nmol/l

What is the most appropriate management?

Refer patient for immediate caesarean section	9%
[✓ Increase carbimazole dose to 20mg once daily]	56%
Commence radioiodine treatment	1%
Switch carbimazole to propylthiouracil	30%
Refer for a thyroidectomy	3%

The carbimazole can be increased to up to 20mg once daily during pregnancy. Propylthiouracil can be started instead of the carbimazole if the increased carbimazole dose does not adequately control the patients hyperthyroidism.

👍 | 👎 | 💬 Discuss (3) | Improve

Next question

Pregnancy: thyroid problems ★

In pregnancy, there is an increase in the levels of thyroxine-binding globulin (TBG). This causes an increase in the levels of total thyroxine but does not affect the free thyroxine level.

Thyrotoxicosis

Untreated thyrotoxicosis increases the risk of fetal loss, maternal heart failure and premature labour

Graves' disease is the most common cause of thyrotoxicosis in pregnancy. It is also recognised that activation of the TSH receptor by HCG may also occur - often termed transient gestational hyperthyroidism. HCG levels will fall in the second and third trimester

Management
- propylthiouracil has traditionally been the antithyroid drug of choice
- however, propylthiouracil is associated with an increased risk of severe hepatic injury
- propylthiouracil is generally used in the first trimester of pregnancy in place of carbimazole, as carbimazole may be associated with an increased risk of congenital abnormalities
- maternal free thyroxine levels should be kept in the upper third of the normal reference range to avoid fetal hypothyroidism
- thyrotrophin receptor stimulating antibodies should be checked at 30-36 weeks gestation - helps to determine the risk of neonatal thyroid problems
- block-and-replace regimes should not be used in pregnancy
- radioiodine therapy is contraindicated

Hypothyroidism

Key points

- thyroxine is safe during pregnancy
- serum thyroid-stimulating hormone measured in each trimester and 6-8 weeks post-partum
- women require an increased dose of thyroxine during pregnancy
    - by up to 50% as early as 4-6 weeks of pregnancy
- breastfeeding is safe whilst on thyroxine

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 23 👎 17

Management of hypothyroidism in pregnancy

Suggest link

Report broken link

Score: **65.5%**

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Question 56 of 101

A 34-year-old man presents to the endocrine clinic for review. He was diagnosed with Addison's disease a year ago, and has found that his treatment of hydrocortisone dosing has restored him to normal function. He no longer feels tired, has regained the weight he lost before becoming unwell, and feels mentally much better. He is now planning a trip to South Africa and was advised to seek advise before travelling. He has already secured a good supply of his hydrocortisone and has obtained medical insurance. What measures should be taken before he travels?

- ○ Advise that malaria prophylaxis is contraindicated whilst taking hydrocortisone ×
- ○ Issue supply of IM hydrocortisone ×
- ○ Advise against travelling to any areas with TB ×
- ○ Advise that he should not fly ×
- ○ Issue supply of prednisolone ×

Submit answer

Reference ranges

Score: **65.5%**

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Question 56 of 101 ✗

A 34-year-old man presents to the endocrine clinic for review. He was diagnosed with Addison's disease a year ago, and has found that his treatment of hydrocortisone dosing has restored him to normal function. He no longer feels tired, has regained the weight he lost before becoming unwell, and feels mentally much better. He is now planning a trip to South Africa and was advised to seek advise before travelling. He has already secured a good supply of his hydrocortisone and has obtained medical insurance. What measures should be taken before he travels?

	Advise that malaria prophylaxis is contraindicated whilst taking hydrocortisone	8%
🟩	Issue supply of IM hydrocortisone	64%
🟥	Advise against travelling to any areas with TB	22%
	Advise that he should not fly	0%
	Issue supply of prednisolone	5%

Patients with Addison's should be given a hydrocortisone injection kit for adrenal crises

Important for me Less important

All patients with Addison's disease should have a supply of IM hydrocortisone and be advised to use this if unable to take oral hydrocortisone or vomiting. This can prevent Addisonian crisis and is especially important when the patient is travelling. It can also be important to supply the patient with the necessary documentation as they will be travelling with needles.

Hydrocortisone does not preclude anti-malarial treatment, and there is no reason that the patient would be unable to fly. It is important to advise the patient to visit a travel clinic to review the level of malaria in the area that they are travelling to, and also to review vaccination status and the need for additional vaccinations.

👍 👎 Discuss (1) Improve

Next question

Addison's disease: management ★

Patients who have Addison's disease are usually given both glucocorticoid and mineralocorticoid replacement therapy.

This usually means that patients take a combination of:
- hydrocortisone: usually given in 2 or 3 divided doses. Patients typically require 20-30 mg per day, with the majority given in the first half of the day
- fludrocortisone

Patient education is important:
- emphasise the importance of not missing glucocorticoid doses
- consider MedicAlert bracelets and steroid cards
- patients should be provided with hydrocortisone for injection with needles and syringes to treat an adrenal crisis
- discuss how to adjust the glucocorticoid dose during an intercurrent illness (see below)

Management of intercurrent illness
- in simple terms the glucocorticoid dose should be doubled, with the fludrocortisone dose staying the same
- the Addison's Clinical Advisory Panel have produced guidelines detailing particular scenarios - please see the CKS link for more details

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries Addison's disease	👍 19	👎 16
Royal College of Physicians 2017 Adrenal insufficiency â€“ recognition and management	👍 16	👎 8

Suggest link

Report broken link

Media

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube

👍 1 👎 0

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **64.3%**

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56 ✗

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A 50-year-old female is admitted with epigastric pain radiating to her back. She has no past medical history and takes no regular medications. Her alcohol history includes 4 glasses of wine per week.

Blood results are as follows:

Hb	115 g/l	Na$^+$	138 mmol/l
Platelets	288 * 10^9/l	K$^+$	3.8 mmol/l
WBC	10.8 * 10^9/l	Urea	6.8 mmol/l
Neuts	8.8 * 10^9/l	Creatinine	72 µmol/l
Lymphs	1.8 * 10^9/l	CRP	84 mg/l
Triglyceride	12.3 mmol/l (normal range < 2.3 mmol/L)	Bilirubin	16 µmol/l
ALT	24 u/l	Amylase	924 U/L
ALP	76 u/l	Albumin	38 g/l

An abdominal ultrasound is performed:

Abdominal ultrasound report	Np gall stones identified. No intra or extra hepatic duct dilation evident

What treatment will reduce the risk of pancreatitis reoccurrence?

○ Ursodeoxycholic acid ✕

○ Fenofibrate ✕

○ Simvastatin ✕

○ Nicotinic acid ✕

○ Laparoscopic cholecystectomy ✕

Submit answer

Reference ranges

Score: **64.3%**

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PassMedicine

← Question 57 of 101 ✗ ⚑ →

A 50-year-old female is admitted with epigastric pain radiating to her back. She has no past medical history and takes no regular medications. Her alcohol history includes 4 glasses of wine per week.

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ALT	24 u/l	Amylase	924 U/L
ALP	76 u/l	Albumin	38 g/l

An abdominal ultrasound is performed:

Abdominal ultrasound report	Np gall stones identified. No intra or extra hepatic duct dilation evident

What treatment will reduce the risk of pancreatitis reoccurrence?

	Ursodeoxycholic acid	5%
🟩	Fenofibrate	78%
🟥	Simvastatin	11%
	Nicotinic acid	4%
	Laparoscopic cholecystectomy	2%

Fibrates are the most effective drug for treating hypertriglyceridaemia

Important for me Less important

The patient has clinical features of pancreatitis. The raised amylase confirms the diagnosis. The two most common causes of pancreatitis in the UK are excess alcohol and gallstones.

The normal liver function tests and ultrasound rule out gallstone disease as the cause. Therefore a laparoscopic cholecystectomy or treatment with ursodeoxycholic acid is not indicated.

The patient drinks 4 glasses of wine per week. A 250ml glass of wine at 12% alcohol contains 250 * 0.12 = 30 ml of pure alcohol. 10 ml of pure alcohol = 1 unit. Therefore each glass of wine contains 3 unit. Her alcohol intake per week is therefore 12 units which is within the recommended intake. It is unlikely that alcohol is the cause.

A patient has a significantly high triglyceride level. While gallstones and alcohol account for roughly 80% of cases of pancreatitis, hypertriglyceridemia is the next most common aetiology, accounting for roughly 1-4% of cases. The exact mechanism is unclear but it is thought to involve increased concentrations of chylomicrons in the blood. These low-density particles are very large and may obstruct capillaries leading to local ischemia and acidaemia. This local damage can expose triglycerides to pancreatic lipases.

Fibrates, statins, and nicotinic acid will all lower triglyceride levels, however, fibrates are the most effective drug in doing so.

Discuss (2) Improve

Next question

Primary hypertriglyceridaemia

Usually due to polygenic factors

May also be due to lipoprotein lipase deficiency

Management
- fibrates are generally used first-line
- statins do reduce triglyceride levels and they may be indicated, particularly if there is mixed hyperlipidaemia

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **63.2%**

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A 16-year-old male attends the clinic with steatorrhoea and poor growth. He has a family history of abetalipoproteinemia. He is on no regular medications. On examination you note ataxia and reduced visual acuity (6/12 in the right eye and 6/15 in the left eye).

Investigations are as follows:

Hb	110 g/l	Na^+	136 mmol/l
Platelets	380 * 10^9/l	K^+	3.9 mmol/l
WBC	10.1 * 10^9/l	Urea	6.6 mmol/l
Neuts	6.8 * 10^9/l	Creatinine	48 µmol/l

Blood film	Acanthocytosis

What management will you advise?

- ○ Reassurance ×
- ○ Simvastatin ×
- ○ Dietary restriction of fats ×
- ○ Ezetimibe ×
- ○ Increase fat intake ×

Submit answer

Reference ranges

Score: **63.2%**

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Back to top

Question 58 of 101 ✓

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Investigations are as follows:

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Platelets	380 * 10^9/l	K$^+$	3.9 mmol/l
WBC	10.1 * 10^9/l	Urea	6.6 mmol/l
Neuts	6.8 * 10^9/l	Creatinine	48 µmol/l

Blood film	Acanthocytosis

What management will you advise?

Reassurance	4%
Simvastatin	12%
Dietary restriction of fats	37%
Ezetimibe	33%
Increase fat intake	13%

Treatment of abetalipoproteinemia involves dietary restriction of fats, and high-dose vitamin E therapy

Important for me Less important

Abetalipoproteinemia is a rare genetic condition caused by a mutation in the microsomal triglyceride transfer protein resulting in deficiencies in apolipoproteins. Apolipoproteins are essential in the synthesis and exportation of chylomicrons and VLDL. The end results is malabsorption of dietary fats, cholesterol, and fat soluble vitamin (e.g. vitamins K, A, D and E).

Clinical features include steatorrhoea, poor growth, neurological dysfunction, and impaired vision.

Treatment is with dietary restriction of fats, and high-dose vitamin E therapy.

Discuss (2) | Improve

Next question

Abetalipoproteinemia

Abetalipoproteinemia is a rare autosomal recessive disorder caused by a mutation in the microsomal triglyceride transfer protein. This results in deficiencies in the apolipoproteins B-48 and B-100.

Features
- failure to thrive + developmental delay
- steatorrhoea
- retinitis pigmentosa
- cerebellar signs
- deep tendon reflexes are absent
- acanthocytosis
- hypocholesterolaemia

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **63.8%**

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Question 59 of 101

A 55-year-old male presented to his general practitioner with a 4-month history of sweating, fatigue and daytime tiredness. He attributed his tight rings to 'fluid retention' and has been experiencing worsening headaches and deterioration in his vision.

He was diagnosed with acromegaly and underwent surgery for this condition 1 month ago. He has been feeling well since and has not experienced any new symptoms.

Which of the following investigations would be most useful for monitoring the effect of his therapy?

- ○ MRI pituitary ✕
- ○ Echocardiography ✕
- ○ Growth hormone levels ✕
- ○ Insulin-like growth factor levels ✕
- ○ Oral glucose tolerance test ✕

Submit answer

Reference ranges

Score: **63.8%**

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Back to top

Question 59 of 101 ✗

A 55-year-old male presented to his general practitioner with a 4-month history of sweating, fatigue and daytime tiredness. He attributed his tight rings to 'fluid retention' and has been experiencing worsening headaches and deterioration in his vision.

He was diagnosed with acromegaly and underwent surgery for this condition 1 month ago. He has been feeling well since and has not experienced any new symptoms.

Which of the following investigations would be most useful for monitoring the effect of his therapy?

🟥	MRI pituitary	3%
	Echocardiography	1%
	Growth hormone levels	6%
🟩	Insulin-like growth factor levels	85%
	Oral glucose tolerance test	6%

Insulin-like growth factors (IGF-1) have a long half-life and so is a useful measurement to assess growth hormone secretion and therefore screen for acromegaly and monitor the response to therapy. Serum IGF-1 is the most feasible parameter to assess clinical disease activity, in everyday practice in the outpatient clinic setting. It is monitored every 6 months, and growth hormone (GH) levels are done yearly.

Note: Oral glucose tolerance test (plus GH levels) is not helpful for patients receiving somatostatin analogues, and for patients receiving GH receptor antagonist therapy, only IGF-1 should be measured.

👍 👎 Discuss (2) Improve

Next question

Acromegaly: management ★

Trans-sphenoidal surgery is the first-line treatment for acromegaly in the majority of patients.

If a pituitary tumour is inoperable or surgery unsuccessful then medication may be indicated:

- somatostatin analogue
  - directly inhibits the release of growth hormone
  - for example octreotide
  - effective in 50-70% of patients
- pegvisomant
  - GH receptor antagonist - prevents dimerization of the GH receptor
  - once daily s/c administration
  - very effective - decreases IGF-1 levels in 90% of patients to normal
  - doesn't reduce tumour volume therefore surgery still needed if mass effect
- dopamine agonists
  - for example bromocriptine
  - the first effective medical treatment for acromegaly, however now superseded by somatostatin analogues
  - effective only in a minority of patients

External irradiation is sometimes used for older patients or following failed surgical/medical treatment

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

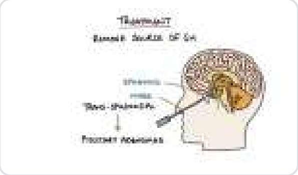
Postgraduate Medical Journal 👍 11 👎 4

Modern treatment of acromegaly

Suggest link

Report broken link

Media



Understanding Acromegaly

Zero To Finals - YouTube 👍 7 👎 1

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **62.7%**

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A 29-year-old woman is referred to the endocrinology clinic. She was concerned about the loss of her period with a negative pregnancy test. She underwent blood tests which demonstrated an undetectable TSH and a free T4 of 48ng/dl. She has a past medical history of hepatitis C but without chronic liver disease. Further tests demonstrate a negative thyroid-stimulating hormone receptor antibodies and a radionuclide thyroid uptake scan demonstrates diffuse thyroid uptake. She is diagnosed with Graves' disease. Due to the previous history of hepatitis C she opts for radioiodine treatment. She is warned regarding avoiding children during treatment, but wonders about her own conception following treatment. What is the most appropriate advise?

- ○ Any future pregnancy will have increased risk of foetal malformation ✕
- ○ She should avoid becoming pregnant for six months after treatment ✕
- ○ She should avoid becoming pregnant for four months after treatment ✕
- ○ Fertility may be adversely affected due to treatment ✕
- ○ She should always avoid breastfeeding ✕

Submit answer

Reference ranges

Score: **62.7%**

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Any future pregnancy will have increased risk of foetal malformation	2%
She should avoid becoming pregnant for six months after treatment	87%
She should avoid becoming pregnant for four months after treatment	8%
Fertility may be adversely affected due to treatment	2%
She should always avoid breastfeeding	1%

Following radioiodine treatment, patients should avoid becoming pregnant for at least 6 months

Important for me Less important

This patient has a diagnosis of Graves' disease based on the biochemical evidence of hyperthyroidism and the diffuse radioiodine uptake scan. Radioiodine is an appropriate treatment choice, but in the absence of severe hepatic dysfunction carbimazole can also be used.

During radioiodine treatment and for three weeks following the treatment, she should avoid contact with small children and pregnant women. She should avoid becoming pregnant for six months following treatment. Following this, the radioiodine carries no risk for her foetus or children. A man on radioiodine treatment should avoid fathering a child for up to four months following treatment. It is important that Graves' disease is biochemically controlled during pregnancy as thyroid-stimulating hormone receptor antibodies can cross the placenta.

👍 👎 Discuss (6) Improve

Next question

Radioiodine therapy ★

Nearly all the iodine ingested is taken up by the thyroid gland. It is an essential element for the formation of T3 and T4. Radioiodine therapy involves using radioactive iodine (I-131 isotope). The radiation can destroy the thyroid gland and any other thyroid cells (including cancer cells) that take up iodine, with little effect on the rest of the body.

Indications

- Differentiated thyroid cancer
- Toxic multinodular goitre
- Graves disease refractory to medical management
- Radiation exposure: Potassium iodine has been used to help individuals exposed to radiation by reducing the harmful accumulation of radioactive substances in the thyroid

Method

- TSH is required for iodine uptake by thyroid cells. In order to effectively treat with radioactive iodine, TSH levels have to be increased to stimulate any thyroid cells into taking up the radioactive iodine. This is done by either making patients hypothyroid after stopping thyroid hormone or by treating with recombinant human TSH.

- A capsule of radioactive iodine is ingested. A clear perspex tube is used to transport the capsule into the patient's mouth to avoid skin contact.
- Once the capsule has been ingested the patient can leave the isotopes unit.

Advice post procedure

- After radioactive iodine, patients should be advised to keep babies, children under five, pregnant women and pets at arm's length for two to three weeks
- It is important not to become pregnant for at least six months after radioactive iodine treatment
- Male patients are advised not to cause a pregnancy for six months after radioactive iodine

Contraindications

- Pregnancy
- Breastfeeding
- Active thyroid eye disease (unless providing steroid cover)

Complications

- Thyroid pain
- Hypothyroidism
- Flare of Graves' eye disease

Follow up

- Monitoring of patients thyroid function (hypothyroidism is the commonest complication of radioiodine therapy)
- For thyroid cancers, monitoring of thyroglobulin. If thyroglobulin concentrations are initially elevated in a person diagnosed with thyroid cancer, then it is likely that thyroglobulin can be used as a tumour marker.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **61.7%**

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Question 61 of 101

A 69-year-old lady presents for a follow-up appointment. She had presented 3 months previously under the acute medical take with headaches, sweating, abdominal pain and wild fluctuations in blood pressure. She is currently being followed up by the appropriate surgical team and her symptoms are currently well controlled with medical treatments. On examination today, you note a lump in her anterior neck and you are given the following blood tests:

Calcium (corrected)	3.68 mmol/l
Phosphate	0.38 mmol/l
Vitamin D3	115 nmol/l (75-200 nmol/l)
Parathyroid hormone	19 pmol/l (0.8 - 8.5 pmol/l)

You have referred the patient to endocrine surgeons for neck biopsies and urgent review. Her daughter, who came with her to the consultation) is concerned she may have the same symptoms later in life. What should you offer the daughter?

- ○ Reassurance ×
- ○ Genetic testing for patient and daughter ×
- ○ Offer annual follow up for surveillance ×
- ○ CT abdomen/pelvis with contrast ×
- ○ Serum bone and calcium homeostasis profile ×

Submit answer

Reference ranges

Score: **61.7%**

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Parathyroid hormone	19 pmol/l (0.8 - 8.5 pmol/l)

You have referred the patient to endocrine surgeons for neck biopsies and urgent review. Her daughter, who came with her to the consultation) is concerned she may have the same symptoms later in life. What should you offer the daughter?

Reassurance	17%
[✓ Genetic testing for patient and daughter]	64%
Offer annual follow up for surveillance	7%
CT abdomen/pelvis with contrast	3%
Serum bone and calcium homeostasis profile	8%

The patient describes features consistent with phaeochromocytoma, the appropriate surgical team would be endocrine surgery, who would arrange for a resection. The new blood tests are suggestive of primary hyperparathyroidism, the neck lumps possible medullary thyroid tumour, resulting in a unifying diagnosis of MEN 2a. The daughter can undergo genetic testing for RET-mutation.

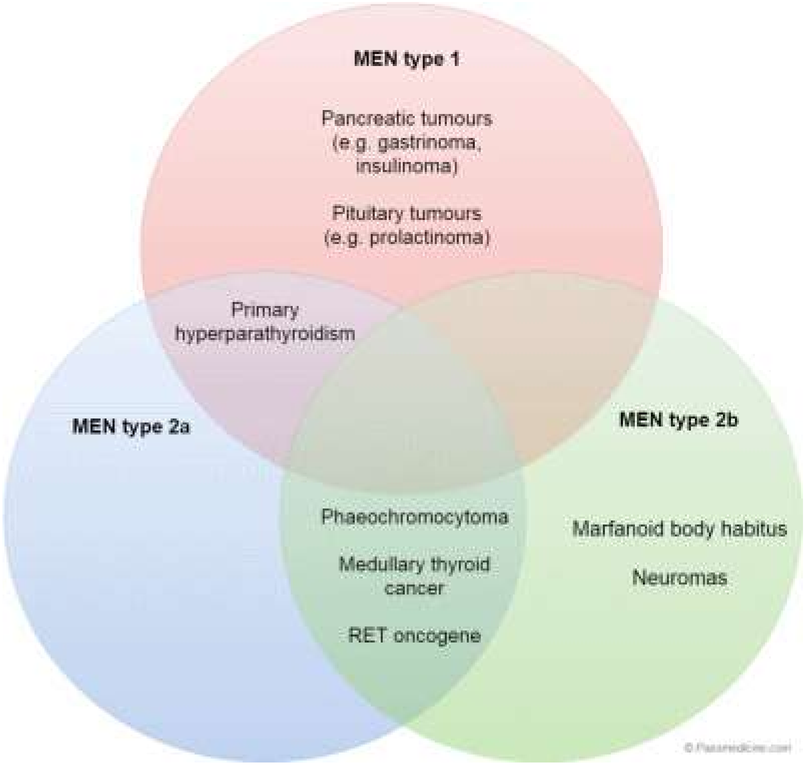
👍 👎 Discuss (5) Improve

Next question

Multiple endocrine neoplasia ★

The table below summarises the three main types of multiple endocrine neoplasia (MEN). MEN is inherited as an autosomal dominant disorder.

MEN type I	**MEN type IIa**	**MEN type IIb**
3 **P**'s **P**arathyroid (95%): hyperparathyroidism due to parathyroid hyperplasia **P**ituitary (70%) **P**ancreas (50%): e.g. insulinoma, gastrinoma (leading to recurrent peptic ulceration) Also: adrenal and thyroid	Medullary thyroid cancer (70%) 2 **P**'s **P**arathyroid (60%) **P**haeochromocytoma	Medullary thyroid cancer 1 **P** **P**haeochromocytoma Marfanoid body habitus Neuromas
MEN1 gene Most common presentation = hypercalcaemia	RET oncogene	RET oncogene


MEN type 1
Pancreatic tumours (e.g. gastrinoma, insulinoma)
Pituitary tumours (e.g. prolactinoma)
Primary hyperparathyroidism
MEN type 2a
MEN type 2b
Phaeochromocytoma
Medullary thyroid cancer
RET oncogene
Marfanoid body habitus
Neuromas
© Passmedicine.com

Venn diagram showing the different types of MEN and their associated features

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PANCREATIC NEUROENDOCRINE NEOPLASMS

Pancreatic neuroendocrine neoplasms

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **62.3%**

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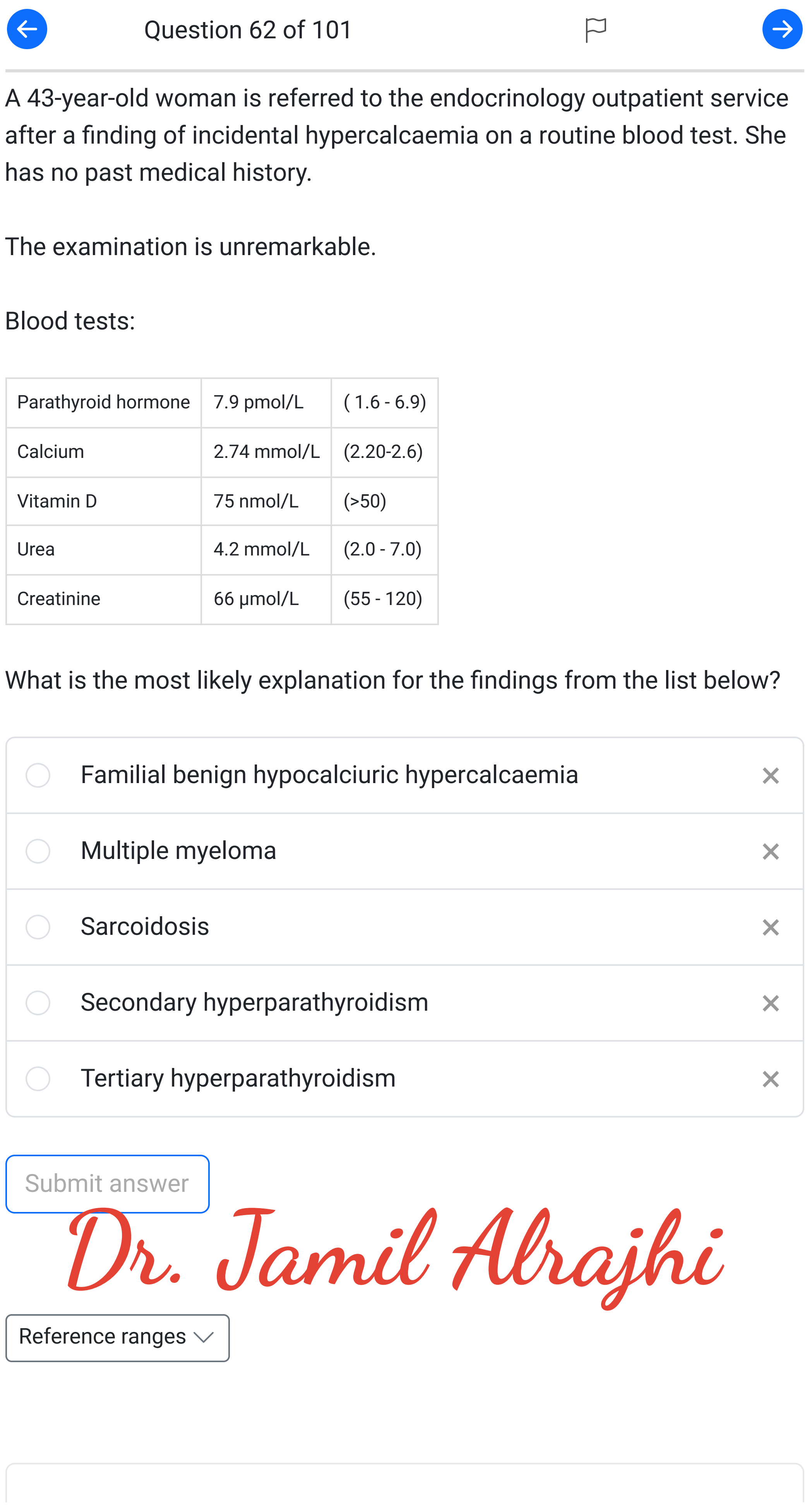

A 43-year-old woman is referred to the endocrinology outpatient service after a finding of incidental hypercalcaemia on a routine blood test. She has no past medical history.

The examination is unremarkable.

Blood tests:

Parathyroid hormone	7.9 pmol/L	(1.6 - 6.9)
Calcium	2.74 mmol/L	(2.20-2.6)
Vitamin D	75 nmol/L	(>50)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)

What is the most likely explanation for the findings from the list below?

- ○ Familial benign hypocalciuric hypercalcaemia ×
- ○ Multiple myeloma ×
- ○ Sarcoidosis ×
- ○ Secondary hyperparathyroidism ×
- ○ Tertiary hyperparathyroidism ×

Dr. Jamil Alrajhi

Score: **62.3%**

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Question 62 of 101 ✓

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The examination is unremarkable.

Blood tests:

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Calcium	2.74 mmol/L	(2.20-2.6)
Vitamin D	75 nmol/L	(>50)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)

What is the most likely explanation for the findings from the list below?

Familial benign hypocalciuric hypercalcaemia	60%
Multiple myeloma	1%
Sarcoidosis	11%
Secondary hyperparathyroidism	14%
Tertiary hyperparathyroidism	14%

Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

Important for me Less important

Familial benign hypocalciuric hypercalcaemia is the correct answer. The patient has hypercalcaemia with a raised PTH level. Plausible differentials include primary hyperparathyroidism, tertiary hyperparathyroidism and familial benign hypocalciuric hypercalcaemia. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Primary hyperparathyroidism is not listed as an option. Therefore, familial benign hypocalciuric hypercalcaemia is the correct answer.

Multiple myeloma is incorrect. Classically, malignancies cause a suppressed PTH level if they are the cause of hypercalcemia. This is true of multiple myeloma.

Sarcoidosis is incorrect. Hypercalcemia in sarcoidosis is due to the uncontrolled production of 1,25-dihydroxy vitamin D3 by macrophages. This causes hypercalcemia and a suppressed PTH. unlike in some forms of hyperparathyroidism, the negative feedback mechanism whereby high calcium causes a low PTH is maintained.

Secondary hyperparathyroidism is incorrect. This form of hyperparathyroidism occurs as an appropriate reaction to low vitamin D or calcium. In this case, the vitamin is normal and the calcium is high.

Tertiary hyperparathyroidism is incorrect. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Therefore this is not likely.

👍 👎 💬 Discuss (6) Improve

Next question

Familial benign hypocalciuric hypercalcaemia

Familial benign hypocalciuric hypercalcaemia is a rare autosomal dominant disorder characterised by asymptomatic hypercalcaemia. It is due to a defect in the calcium-sensing receptor and a decreased sensitivity to increases in extracellular calcium.

The parathyroid hormone level is often not suppressed, as would be expected in all non-hyperparathyrodism related cases of hypercalcaemia. This is due to the decreased sensitivity to increases in extracellular calcium.

+ Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

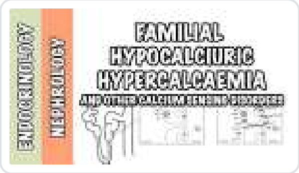
Oxford Academic Website 👍 2 👎 0

Urine Calcium: Laboratory Measurement and Clinical Utility

Suggest link

Report broken link

Media


ENDOCRINOLOGY
NEPHROLOGY
FAMILIAL HYPOCALCIURIC HYPERCALCAEMIA
AND OTHER CALCIUM SENSING DISORDERS

Familial Hypocalciuric Hypercalcemia + other calcium sensing disorders

Armando Hasudungan - YouTube

👍 7 👎 1

Report broken media

Score: **62.9%**

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62 ✓

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Question 63 of 101

A 24-year-old lady presents to hospital with increasing confusion. Her parents describe a gradual history of weight loss, lethargy with abdominal cramping. She has no past medical history and is prescribed no regular medications. She lives with her parents. Her mother suffers from hypothyroidism and her father from hypertension which is controlled with bendroflumethiazide.

On examination she is thin, with cool skin and sunken eyes. Her capillary refill time is 3 seconds with dry mucous membranes. Auscultation of her chest reveals bilateral symmetrical vesicular breath sounds. Her abdomen is soft with normal bowel sounds. She is confused with a Glasgow Coma Scale of 14. She has no focal neurology.

Her investigations reveal;

Hb	10.4 g/dL
MCV	90 fL
WCC	6.4 *10^9/l
Platelets	170 *10^9/l

Na+	105 mmol/L
K+	5.8 mmol/L
Ur	8.8 mmol/L
Cr	90 µmol/L
Glucose	3.9 mmol/L

Urinary Osmolality	108 mmol/L
Urinary Sodium	67 mmol/L

Chest X ray	Clear
CT Head	No intracranial abnormalities

What is the most likely diagnosis?

- ○ Adrenal insufficiency ×
- ○ Laxative abuse ×
- ○ Hypothyroidism ×
- ○ Diuretic use ×
- ○ SiADH ×

Submit answer

Reference ranges ∨

Score: **62.9%**

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Question 63 of 101

A 24-year-old lady presents to hospital with increasing confusion. Her parents describe a gradual history of weight loss, lethargy with abdominal cramping. She has no past medical history and is prescribed no regular medications. She lives with her parents. Her mother suffers from hypothyroidism and her father from hypertension which is controlled with bendroflumethiazide.

On examination she is thin, with cool skin and sunken eyes. Her capillary refill time is 3 seconds with dry mucous membranes. Auscultation of her chest reveals bilateral symmetrical vesicular breath sounds. Her abdomen is soft with normal bowel sounds. She is confused with a Glasgow Coma Scale of 14. She has no focal neurology.

Her investigations reveal;

Hb	10.4 g/dL
MCV	90 fL
WCC	6.4 *10^9/l
Platelets	170 *10^9/l

Na+	105 mmol/L
K+	5.8 mmol/L
Ur	8.8 mmol/L
Cr	90 µmol/L
Glucose	3.9 mmol/L

Urinary Osmolality	108 mmol/L
Urinary Sodium	67 mmol/L

Chest X ray	Clear
CT Head	No intracranial abnormalities

What is the most likely diagnosis?

- ○ Adrenal insufficiency ×
- ○ Laxative abuse ×
- ○ Hypothyroidism ×
- ○ Diuretic use ×
- ○ SiADH ×

Submit answer

Reference ranges ⌄

Score: **62.9%**

1 ✓
2 ✓
3 ×
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5 ×
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7 ×
8 ✓
9 ✓

Dr. Jamil Alrajhi

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Cr	90 µmol/L
Glucose	3.9 mmol/L

Urinary Osmolality	108 mmol/L
Urinary Sodium	67 mmol/L

Chest X ray	Clear
CT Head	No intracranial abnormalities

What is the most likely diagnosis?

[✓ Adrenal insufficiency]	56%
Laxative abuse	5%
Hypothyroidism	2%
Diuretic use	27%
SiADH	10%

This lady has hypovolaemic hyponatraemia. Sodium can be lost through the gastrointestinal tract, skin or urinary tract. This patient is likely to have renal loss secondary to adrenal insufficiency.

The urinary sodium would be reduced if there were gastrointestinal or skin losses. Also, the patient would be hypokalaemic if there were laxative abuse. The patient would be expected to have hypokalaemia if there were abuse of bendrofumethiazide.

The patient is hypovolaemic and therefore SiADH is excluded, also, hypothyroidism and adrenal insufficiency have not been excluded which would be necessary to make the diagnosis of SiADH.

Hypothyroidism classically presents with weight gain and euvolaemic hyponatraemia.

This patient has adrenal insufficiency. It is possibly of an autoimmune nature with a probable autoimmune hypothyroidism in the immediate family. She has a gradual onset of non-specific symptoms lethargy, weight loss and abdominal symptoms. The hyponatraemia has caused confusion. The blood tests reveal hypoglycaemia, hyponatraemia with hyperkalaemia and normocytic anaemia. Examination revealed signs of hypovolaemia, it also could show skin or mucus membrane pigmentation.

👍 👎 Discuss (8) Improve

Next question

Addisonian crisis ★

Causes

- sepsis or surgery causing an acute exacerbation of chronic insufficiency (e.g. Addison's, hypopituitarism)
- adrenal haemorrhage e.g. Waterhouse-Friderichsen syndrome (fulminant meningococcaemia)
- steroid withdrawal (most common precipitant)
- inadequate 'sick day rule' management or intercurrent illness (commonly gastroenteritis) in patients with underlying Addison's disease

Signs and symptoms

- confusion, syncope, lethargy, convulsions
- hypotension, tachycardia, hypothermia
- hyponatraemia, hyperkalaemia, hypoglycaemia
- abdominal pain or cramps

Management

- hydrocortisone 100 mg IM or IV followed by 200 mg per 24 hours as a continuous infusion or 50 mg IV or IM every 6 hours (taper according to response as the patient becomes stable)
- 1 litre of normal saline infused over 30–60 minutes, then a further 4–6 litres over the first 24 hours if required; include dextrose if hypoglycaemic
- no fludrocortisone is required acutely as high-dose cortisol exerts a weak mineralocorticoid effect
- once stable, convert to oral replacement after approximately 24 hours, reducing to maintenance dosing over 3–4 days
- identify and treat the precipitating cause (e.g. infection)

- educate patients (and carers) about 'sick day rules' and encourage use of steroid alert cards or bracelets to prevent future crises

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 8 2

2017 Adrenal insufficiency â€“ recognition and management

Suggest link Report broken link

Media

Primary adrenal insufficiency (Addison's disease)

Osmosis - YouTube 6 0

ADDISON'S DISEASE

Addison's disease

Armando Hasudungan - YouTube 👍 4 👎 0

Report broken media

Score: **63.5%**

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A 43-year-old woman presents to the emergency department with abdominal pain radiating from her right loin to right groin. Her past medical history includes Sjogren's syndrome. She takes regular lubricating eye drops and mouth spray. She does not smoke or drink alcohol.

Her observations are heart rate 94 beats per minute, blood pressure 135/88mmHg, respiratory rate 18/minute, oxygen saturations 97% on air and temperature 36.7°C.

On examination, there is left costovertebral angle tenderness. There is no evidence of peritonism and abdominal sounds are present. The cardio-respiratory examination is unremarkable.

Urinalysis:

Leucocytes	negative
Nitrites	negative
Blood	++
Glucose	-ve
pH	5.7 (<5.3)

Venous blood gas:

pH	7.27	(7.35-7.45)
Lactate	0.8 mmol/L	(0.0-2.0)
HCO3-	15 mmol/L	(22-26)
Cl-	110 mEq/L	(98-106)
Na	135 mmol/L	(135-145)
K+	3.1 mmol/L	(3.5-5.0)

Blood tests:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	3.0 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Calcium	2.05mmol/L	(2.20-2.60)
Parathyroid hormone	7.8 pmol/L	(1.6-6.9)

A CT KUB demonstrates a left-sided ureteric stone but also bilateral nephrocalcinosis.

What is the most likely explanation for the collected findings?

○ Primary hyperparathyroidism ×

○ Tertiary hyperparathyroidism ×

○ Type 1 renal tubular acidosis ×

○ Type 2 renal tubular acidosis ×

○ Type 4 renal tubular acidosis ×

Submit answer

Reference ranges

Score: **63.5%**

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A CT KUB demonstrates a left-sided ureteric stone but also bilateral nephrocalcinosis.

What is the most likely explanation for the collected findings?

Primary hyperparathyroidism	2%
Tertiary hyperparathyroidism	1%
Type 1 renal tubular acidosis	87%
Type 2 renal tubular acidosis	9%
Type 4 renal tubular acidosis	2%

Hypokalaemia, nephrocalcinosis - type 1 renal tubular acidosis

Important for me Less important

Type 1 renal tubular acidosis is the correct answer. This is characterized by failure of hydrogen ion secretion into the lumen of the nephron. It leads to an inability to acidify the pH of the urine to < 5.3. This leads to a normal anion gap metabolic acidosis, hypokalaemia, hyperchloraemia, hypocalcaemia and in some cases, the development of nephrocalcinosis and renal stones. The development of this condition is associated with Sjogren's syndrome. The development of nephrocalcinosis is not seen in other types of renal tubular acidosis.

Type 2 renal tubular acidosis is incorrect. This can cause a normal anion gap metabolic acidosis (NAGMA) and hypokalemia. However, this is more associated with the development of osteomalacia. Nephrocalcinosis does not develop in this condition.

Type 4 renal tubular acidosis is incorrect. This is characterized by a NAGMA and hyperkalemia rather than hypokalaemia. This is associated with drugs such as NSAIDs and diabetic nephropathy.

Primary hyperparathyroidism is incorrect. The PTH level is appropriately raised given the low calcium.

Tertiary hyperparathyroidism is incorrect. This would cause a raised PTH in the setting of raised calcium, typically as a consequence of advanced chronic kidney disease. This is not the biochemical profile seen in this case.

👍 👎 💬 Discuss (5) Improve

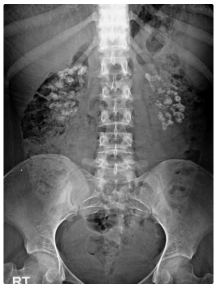
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)
- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones

- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia
- causes include hypoaldosteronism, diabetes

+ Q 123

Next question

Textbooks

High-yield textbook

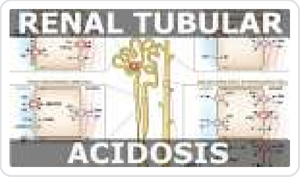
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **64.1%**

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64	✓

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Question 65 of 101

A 70-year-old woman is reviewed in the chronic kidney disease clinic. She also has a history of hypertension for which she takes amlodipine 5mg od and ramipril 10mg od. Her most recent results are as follows:

Blood pressure today is 128/74 mmHg.

	Recent	**12 months ago**
Na$^+$	140 mmol/l	141 mmol/l
K$^+$	4.5 mmol/l	4.3 mmol/l
Urea	11.2 mmol/l	10.5 mmol/l
Creatinine	124 µmol/l	114 µmol/l
eGFR	39 ml/min	43 ml/min

What is the most appropriate next step in management?

- ○ Start atorvastatin 20mg on ×
- ○ Reduce ramipril to 5mg od and recheck U&Es in 4 weeks ×
- ○ Start simvastatin 40mg on ×
- ○ Increase amlodipine to 10mg od ×
- ○ Check her QRISK2 score ×

Submit answer

Reference ranges

Score: **64.1%**

1	✓
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Urea	11.2 mmol/l	10.5 mmol/l
Creatinine	124 µmol/l	114 µmol/l
eGFR	39 ml/min	43 ml/min

What is the most appropriate next step in management?

🟩	Start atorvastatin 20mg on	40%
	Reduce ramipril to 5mg od and recheck U&Es in 4 weeks	16%
	Start simvastatin 40mg on	3%
	Increase amlodipine to 10mg od	1%
🟥	Check her QRISK2 score	40%

QRISK2 should not be used in patients with chronic kidney diseas (CKD). NICE now recommends that all patients with CKD should take a statin.

> *Offer atorvastatin 20 mg for the primary or secondary prevention of*

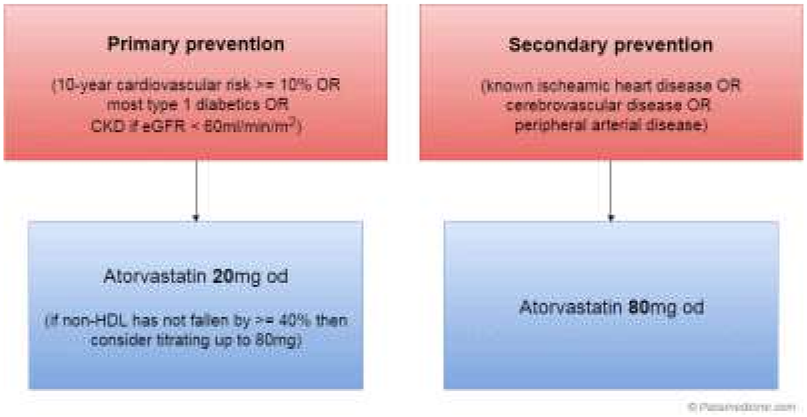
> *CVD to people with CKD*
> - *Increase thedose if a greater than 40% reduction in nonHDL cholesterol is not achieved and eGFR is 30 ml/min*
> - *Agree the use of higher doses with a renal specialist if eGFR is less than 30 ml/min*

Discuss (5) | Improve

Next question

Hyperlipidaemia: management

In 2014 NICE updated their guidelines on lipid modification. This proved highly controversial as it meant that we should be recommending statins to a significant proportion of the population over the age of 60 years. Anyway, the key points of the new guidelines are summarised below.


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
© Passmedicine.com

Graphic showing choice of statin.

Primary prevention - who and how to assess risk

A systematic strategy should be used to identify people aged over 40 years who are likely to be at high risk of cardiovascular disease (CVD), defined as a 10-year risk of **10%** or greater.

NICE recommend we use the **QRISK2** CVD risk assessment tool for patients aged <= 84 years. Patients >= 85 years are at high risk of CVD due to their age. QRISK2 should not be used in the following situations as there are more specific guidelines for these patient groups:

- type 1 diabetics
- patients with an estimated glomerular filtration rate (eGFR) less than 60 ml/min and/or albuminuria
- patients with a history of familial hyperlipidaemia

NICE suggest QRISK2 may underestimate CVD risk in the following population groups:

- people treated for HIV
- people with serious mental health problems
- people taking medicines that can cause dyslipidaemia such as antipsychotics, corticosteroids or immunosuppressant drugs
- people with autoimmune disorders/systemic inflammatory disorders such as systemic lupus erythematosus

Measuring lipid levels

When measuring lipids both the total cholesterol and HDL should be checked to provide the most accurate risk of CVD. A full lipid profile should also be checked (i.e. including triglycerides) before starting a statin. The samples do not need to be fasting.

In the vast majority of patients the cholesterol measurements will be fed into the QRISK2 tool. If however, the patient's cholesterol is very high we should consider familial hyperlipidaemia. NICE recommend the following that we should consider the possibility of familial hypercholesterolaemia and investigate further if:

- the total cholesterol level greater than 7.5 mmol/L and/or
- there is a personal or family history of premature coronary heart disease (an event before 60 years in an index person or first-degree relative [parents, siblings, children])

Interpreting the QRISK2 result

Probably the headline change in the 2014 guidelines was the new, lower cut-off of 10-year CVD risk cut-off of 10%.

NICE now recommend we offer a statin to people with a QRISK2 10-year risk of >= 10%

Lifestyle factors are of course important and NICE recommend that we give patients the option of having their CVD risk reassessed after a period of time before starting a statin.

Atorvastatin 20mg should be offered first-line.

Special situations

Type 1 diabetes mellitus

- NICE recommend that we 'consider statin treatment for the primary prevention of CVD in all adults with type 1 diabetes'
- atorvastatin 20 mg should be offered if type 1 diabetics who are:
  - older than 40 years, or
  - have had diabetes for more than 10 years or
  - have established nephropathy or
  - have other CVD risk factors

Chronic kidney disease (CKD)

- atorvastatin 20mg should be offered to patients with CKD
- increase the dose if a greater than 40% reduction in non-HDL cholesterol is not achieved and the eGFR > 30 ml/min. If the eGFR is < 30 ml/min a renal specialist should be consulted before increasing the dose

Secondary prevention

All patients with CVD should be taking a statin in the absence of any contraindication.

Atorvastatin 80mg should be offered first-line.

Follow-up of people started on statins

NICE recommend we follow up patients at 3 months

- repeat a full lipid profile
- if the non-HDL cholesterol has not fallen by at least 40% concordance and lifestyle changes should be discussed with the patient
- NICE recommend we consider increasing the dose of atorvastatin up to 80mg

Lifestyle modifications

These are in many ways predictable but NICE make a number of specific points:

Cardioprotective diet

- total fat intake should be <= 30% of total energy intake
- saturated fats should be <= 7% of total energy intake
- intake of dietary cholesterol should be < 300 mg/day
- saturated fats should be replaced by monounsaturated and polyunsaturated fats where possible
- replace saturated and monounsaturated fat intake with olive oil, rapeseed oil or spreads based on these oils
- choose wholegrain varieties of starchy food
- reduce their intake of sugar and food products containing refined sugars including fructose
- eat at least 5 portions of fruit and vegetables per day
- eat at least 2 portions of fish per week, including a portion of oily fish
- eat at least 4 to 5 portions of unsalted nuts, seeds and legumes per week

Physical activity

- each week aim for at least 150 minutes of moderate-intensity aerobic activity or 75 minutes of vigorous-intensity aerobic activity or a mix of moderate and vigorous aerobic activity
- do muscle-strengthening activities on 2 or more days a week that work all major muscle groups (legs, hips, back, abdomen, chest, shoulders and arms) in line with national guidance for the general population

Weight management

- no specific advice is given, overweight patients should be managed in keeping with relevant NICE guidance

Alcohol intake

- again no specific advice, other than the general recommendation: men and women are advised not to drink more than 14 units a week on a regular basis

Smoking cessation

- smokers should be encouraged to quit

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 9 👎 16

Managing lipids in type 2 diabetes

NICE 👍 12 👎 14

2014 Lipid modification guidelines

Suggest link Report broken link

Score: **63.1%**

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A 60-year-old man attends a medical health check-up at his GP surgery. He was fit and well with a past medical history of childhood asthma and osteoarthritis in his fingers. His observations were included a blood pressure of 129/80 mmHg, pulse of 82 bpm, and oxygen sats of 97%.

Blood tests were performed and revealed:

Hb	138 g/l	
Platelets	190 * 10^9/l	
WBC	7.6 * 10^9/l	
Na^+	139 mmol/l	
K^+	3.9 mmol/l	
Urea	4.1 mmol/l	
Creatinine	92 µmol/l	
Bilirubin	15 µmol/l	
ALP	52 u/l	
ALT	26 u/l	
γGT	58 u/l	
Albumin	40 g/l	
Serum corrected calcium	2.77 mmol/L	
Serum phosphate	0.90 mmol/l	
Parathyroid hormone	5.9 pmol/L	normal range 1.2-5.8 pmol/L

A 24 hour urinary calcium test was performed based on the results above and revealed a result of 0.5 mmol/24 hours (normal range 2.4-7.4 mmol/24 hours)

What is the most likely diagnosis?

○ Primary hyperparathyroidism ×

○ Secondary hyperparathyroidism ×

○ Vitamin D toxicity ×

○ Multiple endocrine neoplasia type I ×

○ Familial benign hypocalciuric hypercalcaemia ×

Submit answer

Reference ranges ⌄

Score: **63.1%**

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Back to top

A 60-year-old man attends a medical health check-up at his GP surgery. He was fit and well with a past medical history of childhood asthma and osteoarthritis in his fingers. His observations were included a blood pressure of 129/80 mmHg, pulse of 82 bpm, and oxygen sats of 97%.

Blood tests were performed and revealed:

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γGT	58 u/l	
Albumin	40 g/l	
Serum corrected calcium	2.77 mmol/L	
Serum phosphate	0.90 mmol/l	
Parathyroid hormone	5.9 pmol/L	normal range 1.2-5.8 pmol/L

A 24 hour urinary calcium test was performed based on the results above and revealed a result of 0.5 mmol/24 hours (normal range 2.4-7.4 mmol/24 hours)

What is the most likely diagnosis?

Primary hyperparathyroidism	19%
Secondary hyperparathyroidism	2%
Vitamin D toxicity	1%
Multiple endocrine neoplasia type I	1%
Familial benign hypocalciuric hypercalcaemia	77%

Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

Important for me Less important

The most likely diagnosis in this scenario is familial benign hypocalciuric hypercalcaemia. Most cases are asymptomatic and blood test reveals hypercalcaemia with a reduced calcium urinary excretion rate (of under 0.02 mmol/L). There may also be normal to high parathyroid hormone, despite the elevated serum calcium levels.

👍 👎 Discuss (4) Improve

Next question

Familial benign hypocalciuric hypercalcaemia ★

Familial benign hypocalciuric hypercalcaemia is a rare autosomal dominant disorder characterised by asymptomatic hypercalcaemia. It is due to a defect in the calcium-sensing receptor and a decreased sensitivity to increases in extracellular calcium.

The parathyroid hormone level is often not suppressed, as would be expected in all non-hyperparathyrodism related cases of hypercalcaemia. This is due to the decreased sensitivity to increases in extracellular calcium.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

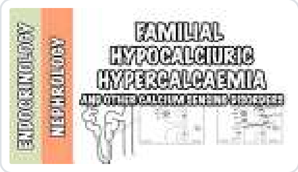
Oxford Academic Website 👍 2 👎 0

Urine Calcium: Laboratory Measurement and Clinical Utility

Suggest link

Report broken link

Media


ENDOCRINOLOGY
NEPHROLOGY
FAMILIAL HYPOCALCIURIC HYPERCALCAEMIA
AND OTHER CALCIUM SENSING DISORDERS

Familial Hypocalciuric Hypercalcemia + other calcium sensing disorders

Armando Hasudungan - YouTube 👍 7 👎 1

Report broken media

Score: **63.6%**

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A 53-year-old obese HGV driver, normally taking BD Novomix 30 insulin presents to your outpatient clinic to clarify some driving regulations he had overheard while eating with colleagues. He is extremely tearful and anxious. He is worried about losing his livelihood as a result of his diabetes.

He was first diagnosed with type 2 diabetes 9 years ago and became insulin dependent 2 years ago. He reports good compliance with insulin every day. However, 18 months ago, he took the same units of insulin after exercising and felt giddy. A spot blood glucose check demonstrated 2.8 mmol/l, which improved immediately after drinking Lucozade that he carried with him. No hospitalisation was required. He has no other past medical history. He has no visual field or peripheral nerve impairments. What is the advice you give him regarding driving?

- ○ Can continue driving, review in 1 year ×
- ○ Can continue driving, no further reviews required ×
- ○ Must stop driving and give up license permanently ×
- ○ Must stop driving temporarily and review in 6 months ×
- ○ Patient can drive type 1 vehicles (cars, motorcycles) but not type 2 vehicles (lorries, HGV) and should reconsider his profession ×

Submit answer

Reference ranges

Score: **63.6%**

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Question 67 of 101 ✕

A 53-year-old obese HGV driver, normally taking BD Novomix 30 insulin presents to your outpatient clinic to clarify some driving regulations he had overheard while eating with colleagues. He is extremely tearful and anxious. He is worried about losing his livelihood as a result of his diabetes.

He was first diagnosed with type 2 diabetes 9 years ago and became insulin dependent 2 years ago. He reports good compliance with insulin every day. However, 18 months ago, he took the same units of insulin after exercising and felt giddy. A spot blood glucose check demonstrated 2.8 mmol/l, which improved immediately after drinking Lucozade that he carried with him. No hospitalisation was required. He has no other past medical history. He has no visual field or peripheral nerve impairments. What is the advice you give him regarding driving?

🟩	Can continue driving, review in 1 year	37%
	Can continue driving, no further reviews required	10%
🟥	Must stop driving and give up license permanently	4%
	Must stop driving temporarily and review in 6 months	15%
	Patient can drive type 1 vehicles (cars, motorcycles) but not type 2 vehicles (lorries, HGV) and should reconsider his profession	33%

The salient points in this case history are that the patient, although insulin dependent for treating his type 2 diabetes, retains hypoglycaemia awareness. A recent change in the DVLA guidelines of May 2012 allows HGV drivers to retain their license even if taking insulin, provided they have not suffered from hypoglycaemia requiring the assistance of others within the past 12 months, and the patient has no visual field impairments. The patient must then be reviewed annually by a diabetes consultant, with 3 months of blood glucose monitoring data available. A 1-year license is then issued annually.

👍 👎 Discuss (2) Improve

Next question

DVLA: diabetes mellitus

Until recently people with diabetes who used insulin could not hold a HGV licence. The DVLA changed the rules in October 2011. The following standards need to be met (and also apply to patients using other hypoglycaemic inducing drugs such as sulfonylureas):

- there has not been any severe hypoglycaemic event in the previous 12 months
- the driver has full hypoglycaemic awareness
- the driver must show adequate control of the condition by regular blood glucose monitoring*, at least twice daily and at times relevant to driving
- the driver must demonstrate an understanding of the risks of hypoglycaemia
- here are no other debarring complications of diabetes

From a practical point of view patients on insulin who want to apply for a Group 2 (HGV) licence need to complete a VDIAB1I form.

Other specific points for group 1 drivers:

- if on insulin then patient can drive a car as long as they have hypoglycaemic awareness, not more than one episode of hypoglycaemia requiring the assistance of another person within the preceding 12 months and no relevant visual impairment. Drivers are normally contacted by DVLA
- if on tablets or exenatide no need to notify DVLA. If tablets may induce hypoglycaemia (e.g. sulfonylureas) then there must not have

been more than one episode of hypoglycaemia requiring the assistance of another person within the preceding 12 months

- if diet controlled alone then no requirement to inform DVLA

*to demonstrate adequate control, the Secretary of State's Honorary Medical Advisory Panel on Diabetes Mellitus has recommended that applicants will need to have used blood glucose meters with a memory function to measure and record blood glucose levels for at least 3 months prior to submitting their application

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

DVLA 👍 7 👎 9

[Fitness to drive guidelines]

Gov.uk 👍 12 👎 4

[Diabetes and driving]

Suggest link Report broken link

Score: **62.7%**

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← Question 68 of 101 →

A 22-year-old woman attended with symptoms of dysuria and increased frequency. A urine dip was positive for leucocytes, nitrites, and glucose. Capillary blood glucose was 11mmol/L. Ketones were normal. She was treated for a urinary tract infection and returned for repeat urine and capillary blood glucose testing 2 weeks later. Her urine sample was again positive for glucose, but negative for leucocytes and nitrites. Her capillary blood glucose was 13. Ketones were normal.

She has a family history of hypertension and her father has a HNF1A genetic mutation.

Given the likely diagnosis, what is the most appropriate initial treatment for the likely diagnosis?

- ○ Dietary and lifestyle change ×
- ○ Insulin ×
- ○ Low-dose gliclazide ×
- ○ Metformin ×
- ○ No treatment required ×

Submit answer

Reference ranges

Score: **62.7%**

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← Question 68 of 101 ✗ ⚐ →

A 22-year-old woman attended with symptoms of dysuria and increased frequency. A urine dip was positive for leucocytes, nitrites, and glucose. Capillary blood glucose was 11mmol/L. Ketones were normal. She was treated for a urinary tract infection and returned for repeat urine and capillary blood glucose testing 2 weeks later. Her urine sample was again positive for glucose, but negative for leucocytes and nitrites. Her capillary blood glucose was 13. Ketones were normal.

She has a family history of hypertension and her father has a HNF1A genetic mutation.

Given the likely diagnosis, what is the most appropriate initial treatment for the likely diagnosis?

	Dietary and lifestyle change	8%
	Insulin	7%
🟩	Low-dose gliclazide	74%
🟥	Metformin	8%
	No treatment required	3%

MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

Important for me Less important

Genetic testing would be required to confirm, but the likely diagnosis here is MODY3 which is associated with HNF1A mutations. These mutations are usually inherited in an autosomal dominant pattern. The presentation is often incidental, with recurrent hyperglycaemia without ketosis.

Low dose gliclazide is the correct answer. HNF1A mutations lead to a reduction in the amount of insulin secreted by the pancreas.

Metformin is incorrect. The main mechanism of action of metformin is increased sensitivity to insulin. Gliclazide is the preferred treatment as the HNF1A mutation reduces insulin production by the pancreas. It responds better to insulin secretagogue treatment than an insulin-sensitising agent.

Insulin is incorrect. Most patients with MODY do not require insulin as there is generally some insulin secretion from the pancreas particularly in early diagnosis. Treatment with a secretagogue such as gliclazide is therefore preferable.

No treatment required is incorrect. All forms of MODY apart from glucokinase MODY increase the risk of long-term complications of diabetes. Treatment to stabilise and control blood glucose levels is therefore advisable.

Dietary advice and lifestyle changes is an incorrect answer. Although this advice would form part of the management, the main treatment would be pharmacological with a low dose of gliclazide. Unlike in early type-2 diabetes or pre-diabetes where there is a possibility of improving insulin sensitivity by decreasing glucose spikes, MODY is not reversible.

👍 👎 💬 Discuss Improve

Next question

MODY ★

Maturity-Onset Diabetes of the Young (MODY) is a form of monogenic diabetes, typically characterized by an autosomal dominant inheritance pattern, onset usually before 25 years of age, and impairment in insulin secretion with minimal or no defects in insulin action. Unlike more common forms of diabetes, such as Type 1 and Type 2, MODY is not primarily driven by lifestyle factors.

It is thought that around 1-2% of patients with diabetes mellitus have MODY, and around 90% are misclassified as having either type 1 or type 2 diabetes mellitus.

Classification and Genetics

MODY is a heterogenous group, with over 14 types identified as of the latest revision, each corresponding to mutations in different genes. The most common types are MODY2 (GCK mutation) and MODY3 (HNF1A mutation), but others include MODY1 (HNF4A), MODY4 (PDX1), MODY5 (HNF1B), and so on.

MODY 3 accounts for around 60% of cases with MODY 2 causing 20% of cases.

Each subtype varies in its clinical presentation, management, and prognosis, emphasizing the importance of precise genetic diagnosis in order to guide treatment strategy and genetic counselling.

Clinical Features

Patients with MODY often present with mild non-ketotic hyperglycemia that is often detected incidentally or during routine screening. It may also be discovered during pregnancy. Unlike Type 1 diabetes, patients with MODY usually do not present with diabetic ketoacidosis except under severe stress conditions, and unlike Type 2 diabetes, they are often of normal weight and do not exhibit signs of insulin resistance.

The specific clinical manifestations and complications can vary depending on the subtype of MODY. For example, individuals with MODY2 generally have mild, stable fasting hyperglycemia and rarely develop severe complications, whereas those with MODY3 or MODY1 may have progressive hyperglycemia and are at higher risk for complications typically associated with diabetes, such as retinopathy, nephropathy, and cardiovascular disease.

Diagnosis

MODY should be suspected in individuals with persistent, asymptomatic hyperglycemia detected before the age of 25, without the typical features of Type 1 or Type 2 diabetes.

The diagnosis is confirmed by genetic testing, which is crucial for identifying the specific type of MODY, as this has direct implications for management. It's important to remember, however, that genetic testing can be expensive and may not be available in all settings.

Treatment

The treatment for MODY depends on the specific genetic subtype. For example, MODY2 often does not require specific treatment, as the hyperglycemia is mild and usually does not lead to complications. In contrast, MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas. Insulin therapy may be necessary in some cases, especially during pregnancy or if sulfonylureas are contraindicated or ineffective.

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Score: **61.8%**

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Back to top

← Question 69 of 101 →

A 29-year-old woman is referred to the Endocrinology clinic as she has just found out she is pregnant. She was diagnosed with hypothyroidism three years ago and is currently stable on a dose of levothyroxine 75mcg od. She has also been taking folic acid 400mcg od for the past 6 months. Her last bloods taken 6 months ago show the following:

TSH	1.4 mU/l

You request a repeat TSH and free T4 measurement. What is the most appropriate next step?

- ○ Decrease levothyroxine to 50mcg od ×
- ○ Keep levothyroxine at 75mcg od ×
- ○ Increase levothyroxine to 100mcg od ×
- ○ Keep levothyroxine at 75mcg od + increase folic acid to 5mg od ×
- ○ Stop levothyroxine until TSH known ×

Submit answer

Reference ranges

Score: **61.8%**

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TSH	1.4 mU/l

You request a repeat TSH and free T4 measurement. What is the most appropriate next step?

Decrease levothyroxine to 50mcg od	2%
Keep levothyroxine at 75mcg od	4%
[✓ Increase levothyroxine to 100mcg od]	85%
Keep levothyroxine at 75mcg od + increase folic acid to 5mg od	9%
Stop levothyroxine until TSH known	0%

Pregnant with hypothyroidism → immediately increase levothyroxine and monitor TSH closely

Important for me Less important

The correct answer is to **increase levothyroxine to 100mcg od**. In pregnant women with hypothyroidism, it is essential to maintain adequate thyroid hormone levels for the developing fetus. The requirement for levothyroxine often increases during pregnancy due to increased thyroxine-binding globulin (TBG) and placental degradation of maternal T4. According to UK guidelines, pregnant women with pre-existing hypothyroidism should have their levothyroxine dose increased by approximately 30% as soon as pregnancy is confirmed, even before obtaining repeat TSH and free T4 measurements. This ensures that both mother and baby have sufficient thyroid hormone levels during the critical early stages of fetal development.

The option to **decrease levothyroxine to 50mcg od** would not be appropriate, as reducing the dose could lead to inadequate thyroid hormone levels in the mother and potentially harm fetal development.

Keeping the **levothyroxine at 75mcg od** might seem reasonable if her TSH was well-controlled prior to pregnancy; however, this does not account for the increased requirements during pregnancy. It would be safer and more appropriate to increase the dose proactively.

As for **keeping levothyroxine at 75mcg od + increasing folic acid to 5mg od**, while it is important for pregnant women to take folic acid supplements, increasing the dose of folic acid alone without adjusting the levothyroxine dosage does not address her increased thyroid hormone needs during pregnancy.

Finally, choosing to **stop levothyroxine until TSH is known** would put both mother and fetus at risk of complications related to untreated or inadequately treated hypothyroidism. It is crucial that patients with pre-existing hypothyroidism continue their levothyroxine therapy during pregnancy, with appropriate dose adjustments as needed.

👍 👎 💬 Discuss (4) Improve

Next question

Hypothyroidism: levothyroxine therapy ★

Key points

- initial starting dose of levothyroxine should be lower in elderly patients and those with ischaemic heart disease. The BNF recommends that for patients with cardiac disease, severe hypothyroidism or patients over 50 years the initial starting dose should be 25mcg od with dose slowly titrated. Other patients should be started on a dose of 50-100mcg od

- following a change in thyroxine dose thyroid function tests should be checked after 8-12 weeks
- the therapeutic goal is 'normalisation' of the thyroid stimulating hormone (TSH) level. As the majority of unaffected people have a TSH value 0.5-2.5 mU/l it is now thought preferable to aim for a TSH in this range
- women with established hypothyroidism who become pregnant should have their dose increased by at least 25-50 micrograms levothyroxine due to the increased demands of pregnancy. The TSH should be monitored carefully, aiming for a low-normal value
- there is no evidence to support combination therapy with levothyroxine and liothyronine

Side-effects of thyroxine therapy

- hyperthyroidism: due to over treatment
- reduced bone mineral density
- worsening of angina
- atrial fibrillation

Interactions

- iron, calcium carbonate
    - absorption of levothyroxine reduced, give at least 4 hours apart

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 15 👎 24

Management of hypothyroidism

Suggest link

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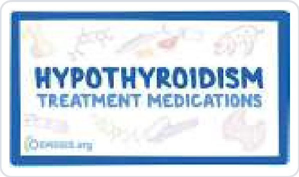
Media


HYPOTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hypothyroidism treatment medications

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **62.3%**

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← Question 70 of 101 →

A 58 year-old man with type 2 diabetes mellitus presents to his GP for his annual review. His diabetes is currently being treated with metformin 500mg twice daily and gliclazide 40mg once daily. He complains that recently he has had several episodes of hypoglycaemia and this has been affecting him at work where his job is as a construction worker. His most recent HbA1c was 66 mmol/mol. Apart from the diabetes, he also has a past medical history of stage 2 heart failure, hypertension and mild chronic obstructive pulmonary disease.

What is the most appropriate treatment change?

- ○ Stop gliclazide and start exenatide ✕
- ○ Stop gliclazide and start sitagliptin ✕
- ○ Commence daily insulin ✕
- ○ Stop gliclazide and start glibenclamide ✕
- ○ Stop gliclazide and start pioglitazone ✕

Submit answer

Reference ranges

Score: **62.3%**

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Question 70 of 101 ✗

A 58 year-old man with type 2 diabetes mellitus presents to his GP for his annual review. His diabetes is currently being treated with metformin 500mg twice daily and gliclazide 40mg once daily. He complains that recently he has had several episodes of hypoglycaemia and this has been affecting him at work where his job is as a construction worker. His most recent HbA1c was 66 mmol/mol. Apart from the diabetes, he also has a past medical history of stage 2 heart failure, hypertension and mild chronic obstructive pulmonary disease.

What is the most appropriate treatment change?

🟥	Stop gliclazide and start exenatide	16%
🟩	Stop gliclazide and start sitagliptin	73%
	Commence daily insulin	3%
	Stop gliclazide and start glibenclamide	3%
	Stop gliclazide and start pioglitazone	5%

This patient's hypoglycaemic attacks is most likely due to the sulfonylurea he is currently on. NICE guidance recommends that if the patient is suffering hypoglycaemic side effects from the sulfonylurea they can be commenced on a combination of metformin and a glitazone or metformin and a DPP-4 inhibitor. However, due to this patient's heart failure, pioglitazone is not suitable, therefore a combination of metformin and sitagliptin is the most appropriate option. Glibenclamide has long-acting hypoglycaemic effects so would not be appropriate in this scenario.

Discuss (8) | Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice

- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation

- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

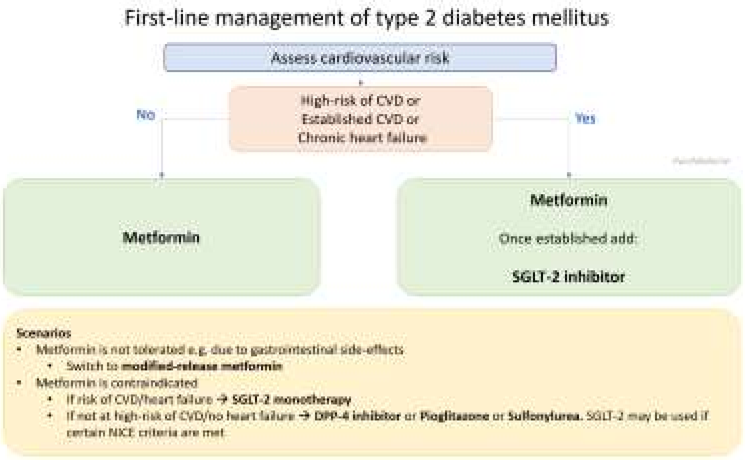
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or Established CVD or Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor
Scenarios
Metformin is not tolerated e.g. due to gastrointestinal side-effects
Switch to modified-release metformin
Metformin is contraindicated
If risk of CVD/heart failure → SGLT-2 monotherapy
If not at high-risk of CVD/no heart failure → DPP-4 inhibitor or Pioglitazone or Sulfonylurea. SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

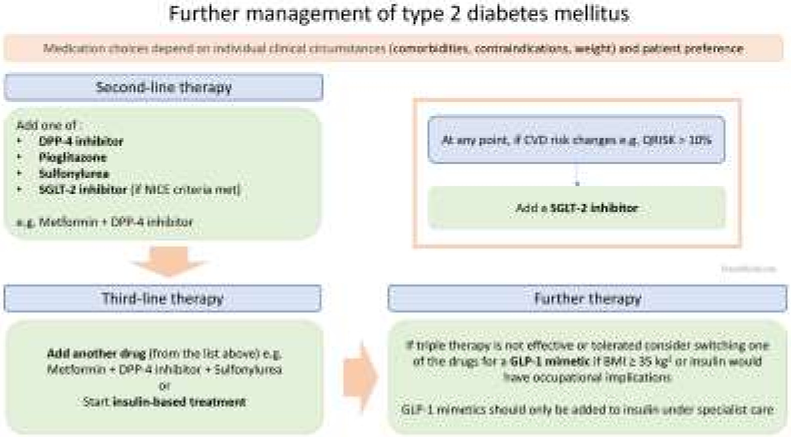
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:

    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
• DPP-4 inhibitor
• Pioglitazone
• Sulfonylurea
• SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg² or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone
- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:

- metformin + DPP-4 inhibitor + sulfonylurea

- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

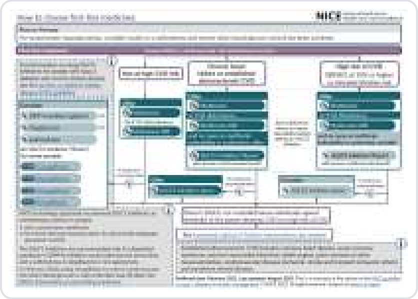
Further therapy

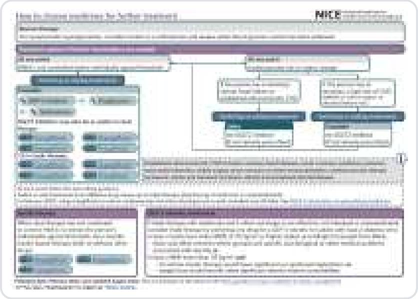
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

GLP-1 mimetics should only be added to insulin under specialist care

Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples
- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea





Risk factor modification

Hypertension

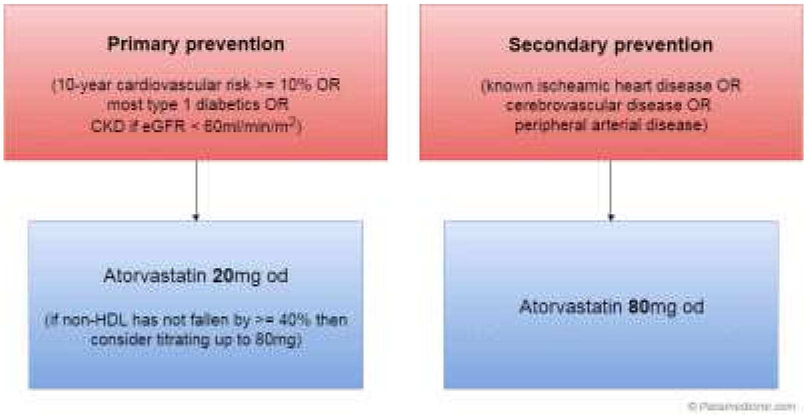
- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line

    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets
- should not be offered unless a patient has existing cardiovascular disease

Lipids
- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m^2)
Secondary prevention
(known ischaemic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
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Graphic showing choice of statin.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

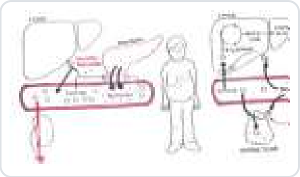
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

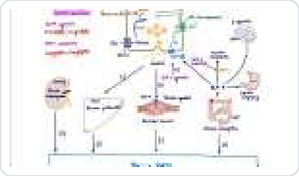
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

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Score: **61.4%**

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Question 71 of 101

A 67-year-old man presents to the GP with a six-month history of excessive tiredness, feeling thirsty, weight loss and passing a lot of urine. He has a strong family history of Type 2 diabetes mellitus.

Examination showed blood pressure of 125/72 mmHg and urinalysis showed ++glucose but no ketones. The abdomen was soft and tender with no masses. His body mass index is 32 kg/m². He is retired and does not drive.

Blood tests arranged showed HbA1c at 58 mmol/mol.

Which of the following is an important part of the management of this patient with new-onset Type 2 Diabetes Mellitus in this age group as per NICE guidelines?

- ○ Referral to secondary care diabetic team for medication review ×
- ○ CT abdomen ×
- ○ No need to discuss medications but repeat bloods in two to three months to monitor ×
- ○ Start gliclazide and titrate to maximum tolerated dose ×
- ○ Issue blood sugar monitoring devices for home checks ×

Submit answer

Reference ranges

Score: **61.4%**

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Blood tests arranged showed HbA1c at 58 mmol/mol.

Which of the following is an important part of the management of this patient with new-onset Type 2 Diabetes Mellitus in this age group as per NICE guidelines?

🟥	Referral to secondary care diabetic team for medication review	37%
🟩	CT abdomen	13%
	No need to discuss medications but repeat bloods in two to three months to monitor	9%
	Start gliclazide and titrate to maximum tolerated dose	15%
	Issue blood sugar monitoring devices for home checks	26%

Patients over the age of 60 with new onset diabetes and weight loss should be referred for an urgent CT abdomen to exclude pancreatic cancer

Important for me Less important

This patient has a new diagnosis of type 2 diabetes mellitus on the basis of symptoms and blood tests. As he is over 60 and has new-onset diagnosis, he should have an urgent CT abdomen performed in order to rule out pancreatic cancer as a cause for the new-onset diabetes in this age group. There is no need to refer to secondary care as metformin can be initiated in primary care. One should consider medications given there is enough in the history and bloods to diagnose type 2 diabetes mellitus - of course, the patient can elect for lifestyle changes if counselled. Gliclazide is not as suitable as metformin in this patient, given his body mass index. One would not routinely give type 2 diabetes mellitus patients blood sugar monitoring devices as Hba1c monitoring is sufficient - an exception would be if the patient is started on a drug that can cause hypoglycaemia for instance.

👍 👎 Discuss (1) Improve

Next question

Diabetes mellitus (type 2): diagnosis

The diagnosis of type 2 diabetes mellitus can be made by either a plasma glucose or a HbA1c sample. Diagnostic criteria vary according to whether the patient is symptomatic (polyuria, polydipsia etc) or not.

If the patient is symptomatic:
- fasting glucose greater than or equal to 7.0 mmol/l
- random glucose greater than or equal to 11.1 mmol/l (or after 75g oral glucose tolerance test)

If the patient is asymptomatic the above criteria apply but must be demonstrated on two separate occasions.

When HbA1c is used for the diagnosis of diabetes:
- a HbA1c of greater than or equal to 48 mmol/mol (6.5%) is diagnostic of diabetes mellitus
- a HbAlc value of less than 48 mmol/mol (6.5%) does not exclude diabetes (i.e. it is not as sensitive as fasting samples for detecting diabetes)

- in patients without symptoms, the test must be repeated to confirm the diagnosis
- it should be remembered that misleading HbA1c results can be caused by increased red cell turnover (see below)

Conditions where HbA1c may not be used for diagnosis:

- haemoglobinopathies
- haemolytic anaemia
- untreated iron deficiency anaemia
- suspected gestational diabetes
- children
- HIV
- chronic kidney disease
- people taking medication that may cause hyperglycaemia (for example corticosteroids)

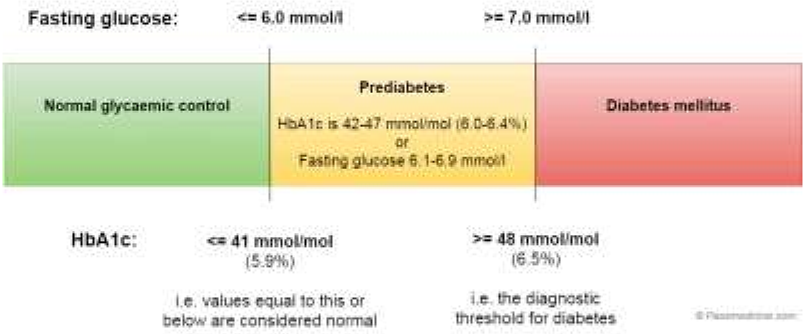

Fasting glucose:
<= 6.0 mmol/l
>= 7.0 mmol/l
Normal glycaemic control
Prediabetes
HbA1c is 42-47 mmol/mol (6.0-6.4%)
or
Fasting glucose 6.1-6.9 mmol/l
Diabetes mellitus
HbA1c:
<= 41 mmol/mol
(5.9%)
i.e. values equal to this or below are considered normal
>= 48 mmol/mol
(6.5%)
i.e. the diagnostic threshold for diabetes

Diagram showing the spectrum of diabetes diagnosis

Impaired fasting glucose and impaired glucose tolerance

A fasting glucose greater than or equal to 6.1 but less than 7.0 mmol/l implies impaired fasting glucose (IFG)

Impaired glucose tolerance (IGT) is defined as fasting plasma glucose less than 7.0 mmol/l and OGTT 2-hour value greater than or equal to 7.8 mmol/l but less than 11.1 mmol/l

Diabetes UK suggests:

- 'People with IFG should then be offered an oral glucose tolerance test to rule out a diagnosis of diabetes. A result below 11.1 mmol/l but above 7.8 mmol/l indicates that the person doesn't have diabetes but does have IGT.'

+ Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 18 👎 20

Type 2 diabetes mellitus: diagnosis

Diabetes UK 👍 23 👎 12

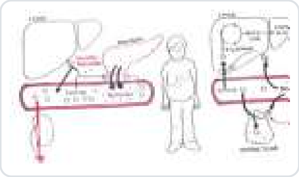
New diagnostic criteria for diabetes

NICE 👍 21 👎 10

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 0 👎 0

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Score: **60.6%**

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A 35-year-old woman presents to the emergency department complaining of shortness of breath and feeling unwell. She has a past medical history of type 1 diabetes mellitus. She is poorly compliant with insulin and has an erratic lifestyle.

Observations:

- Heart rate 89 beats per minute
- Blood pressure 111/77 mmHg
- Oxygen saturations 96% on room air
- Respiratory rate 24/minute
- Temperature 37C

VBG:

pH	7.05	(7.35-7.45)
Lactate	2.8 mmol/L	(0.0-2.0)
HCO3-	8 mmol/L	(22-26)
K+	4.2 mmol/L	(3.5-5.5)
Na+	128 mmol/L	(135-145)
Glucose	31 mmol/L	(4.0-7.8)

Ketones	4.2 mmol/L	(<0.6)

She is commenced on IV fluids (IV 0.9% sodium chloride with added potassium) and a fixed rate insulin infusion.

Repeat investigations four hours post-treatment:

VBG:

pH	7.12	(7.35-7.45)

Lactate	2.2 mmol/L	(0.0-2.0)
HCO3-	12 mmol/L	(22-26)
K+	4.6 mmol/L	(3.5-5.5)
Na+	131 mmol/L	(135-145)
Glucose	10 mmol/L	(4.0-7.8)

Blood ketones	3.2 mmol/L	(<0.6)

Given the likely diagnosis, what is the most appropriate management choice at this point?

- ○ Continue current management and add IV dextrose ×
- ○ No changes to current management ×
- ○ Stop FRII and continue 0.9% sodium chloride with added potassium ×
- ○ Stop current management and switch to IV dextrose ×
- ○ Switch to VRII, continue current IV fluids and add IV dextrose ×

Submit answer

Reference ranges

Score: **60.6%**

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Dr. Jamil Alrajhi

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K+	4.6 mmol/L	(3.5-5.5)
Na+	131 mmol/L	(135-145)
Glucose	10 mmol/L	(4.0-7.8)

Blood ketones	3.2 mmol/L	(<0.6)

Given the likely diagnosis, what is the most appropriate management choice at this point?

🟩	Continue current management and add IV dextrose	73%
	No changes to current management	8%
🟥	Stop FRII and continue 0.9% sodium chloride with added potassium	3%
	Stop current management and switch to IV dextrose	3%
	Switch to VRII, continue current IV fluids and add IV dextrose	13%

DKA in the acute setting fixed rate insulin should be continued even if BM levels are <14; IV dextrose should be added instead

Important for me Less important

Continue FRII and add IV dextrose is correct. The patient has not yet had a resolution of ketonaemia or acidosis. The underlying pathophysiology of DKA is insulin deficiency. Without insulin, the body cannot use glucose for energy and therefore fat sources are broken down to use as fuel, prompting the generation of ketones, which in turn cause acidosis. To reverse this process and resolve ketonemia and acidosis, we need to continue insulin. Insulin therapy causes a lowering of blood glucose levels and therefore we should administer IV dextrose alongside insulin in order to prevent hypoglycaemia while allowing continued insulin administration.

No changes to current management is incorrect. Insulin drives glucose into cells and lowers blood glucose levels. We need to continue insulin until the ketonemia has resolved. However, this will cause hypoglycaemia unless we add IV dextrose.

Stop FRII and continue 0.9% sodium chloride with added potassium is incorrect. While this will result in the resolution of dehydration and avoid precipitating hypoglycaemia (as insulin has been stopped), it will not correct the underlying pathophysiological process (insulin deficiency) and the DKA will worsen.

Stop current management and switch to IV dextrose is not the right answer. Stopping insulin will resulting in worsening DKA, compounded by exogenous glucose administration.

Switch to VRII, continue current IV fluids and add IV dextrose is incorrect. It is standard practice now to administer a fixed rate insulin infusion in DKA rather than a variable rate as an FRII has been proven to be superior as it has been shown to have the following benefits:

- Faster resolution of DKA
- Avoids titration against a poor surrogate marker of blood glucose
- Confirms resolution of DKA if the FRII is stopped when blood ketone levels are < 0.6mmol/L.

👍 👎 💬 Discuss (3) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management
- fluid replacement
    - most patients with DKA are deplete around 5-8 litres

    - isotonic saline is used initially, even if the patient is severely acidotic
    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	**Potassium replacement in mmol/L of infusion solution**
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism

- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

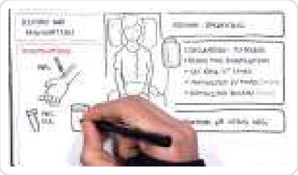
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

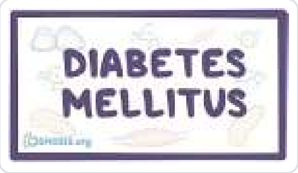
Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2


DIABETES
MELLITUS
osmosis.org

Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

Report broken media

Score: **59.7%**

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← Question 73 of 101 →

A 24-year-old nurse presents after collapsing on a night shift. His blood glucose is measured at being 1.4 mmol/l. His blood pressure at the time was noted to be 115/82 mmHg. He has no palpitations and had not bitten his tongue or become incontinent during the episodes. He was shaken afterwards, although did not have memory loss and stated he had not tripped over anything. He also said he has had five of these episodes over the last two weeks.

Blood tests are sent off and unremarkable except for a low-normal C-peptide level and markedly raised insulin level.

Which of the following is the most likely diagnosis of his multiple episodes of collapse?

- ○ Sulphonylurea misuse ✕
- ○ Insulin misuse ✕
- ○ Alcohol misuse ✕
- ○ Retroperitoneal sarcoma ✕
- ○ Insulinoma ✕

Submit answer

Reference ranges

Score: **59.7%**

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← Question 73 of 101 ✗ ⚑ →

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Blood tests are sent off and unremarkable except for a low-normal C-peptide level and markedly raised insulin level.

Which of the following is the most likely diagnosis of his multiple episodes of collapse?

🟥	Sulphonylurea misuse	7%
🟩	Insulin misuse	85%
	Alcohol misuse	1%
	Retroperitoneal sarcoma	0%
	Insulinoma	6%

Hyperinsulinaemia in the absence of raised C-peptide points towards the diagnosis of insulin abuse. Elevation of C-peptide, when combined with hyperinsulinaemia suggests sulphonylurea abuse. To rule this out it may be appropriate to assay levels of commonly used sulphonylureas in urine. Insulinomas are a more rare cause of repeated hypoglycaemic episodes.

👍 👎 Discuss (5) Improve

Next question

Hypoglycaemia ★

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
    - causes exaggerated insulin secretion
    - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
- nesidioblastosis - beta cell hyperplasia

Physiological response to hypoglycaemia

- hormonal response: the first response of the body is decreased insulin secretion. This is followed by increased glucagon secretion. Growth hormone and cortisol are also released but later
- sympathoadrenal response: increased catecholamine-mediated (adrenergic) and acetylcholine-mediated (cholinergic) neurotransmission in the peripheral autonomic nervous system and in the central nervous system

Features

- blood glucose levels and the severity of symptoms are not always correlated, especially in patients with diabetes.
- blood glucose concentrations <3.3 mmol/L cause autonomic symptoms due to the release of glucagon and adrenaline (average frequency in brackets):
    - Sweating
    - Shaking
    - Hunger
    - Anxiety
    - Nausea

- blood glucose concentrations below <2.8 mmol/L cause neuroglycopenic symptoms due to inadequate glucose supply to the brain:
    - Weakness
    - Vision changes
    - Confusion
    - Dizziness
- Severe and uncommon features of hypoglycaemia include:
    - Convulsion
    - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.

- in the community (for example, diabetes mellitus patients who inject insulin):
    - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form
    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.
    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
    - If the patient is alert, a quick-acting carbohydrate may be given (as above)
    - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
    - Alternatively, intravenous 20% glucose solution may be given through a large vein

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **58.9%**

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A 23-year-old woman is brought into the emergency department by ambulance with her mum, who found her unconscious on the bathroom floor just half an hour ago. The mum had last seen her 4 hours previously and reports she was well at this time. She has a past medical history of depression and was last admitted 5 months ago with a paracetamol overdose. She has otherwise been well in herself and suffers no other medical conditions that mum is aware of. Her GCS on arrival is 8. She is found to be severely hypoglycaemic at 2.2mmol/L and is treated with IV dextrose.

You are the medical registrar on call and have been asked to assess her, she has now been on a dextrose infusion for 45 minutes with no minimal improvement. The examination is grossly normal besides a tachycardia of 110bpm and GCS of 10/15 (M4, E3, V3). She is maintaining her own airways. Up-to-date glucose is 2.9 mmol/L, and her C-peptide and insulin are both elevated. You ask that she be given 50mL of 50% glucose IV, but she remains hypoglycaemic despite this.

Which of the following should be considered as a treatment for this woman?

○ Activated charcoal ×

○ Cyproheptadine ×

○ Dialysis ×

○ Gliclazide ×

○ Octreotide ×

Submit answer

Reference ranges

Score: **58.9%**

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Question 74 of 101 ✓

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Which of the following should be considered as a treatment for this woman?

Activated charcoal	2%
Cyproheptadine	6%
Dialysis	7%
Gliclazide	1%
Octreotide	83%

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Important for me Less important

At this degree of hypoglycaemia, insulin levels should be undetectable. An insulin overdose would only cause raised insulin level; C-peptide is indicative of endogenous insulin release. Therefore the raised levels of both C-peptide and insulin suggest either an insulinoma or sulfonylurea use (triggering endogenous insulin release). Considering her past medical history, sulfonylurea overdose should be considered the most likely cause. Though it is unlikely that she is taking a sulfonylurea, it is quite possible that a family member is taking this medication, and that she might have easy access to it.

An overdose of sulphonylureas such as gliclazide can lead to severe and prolonged hypoglycaemia. The elimination half-life of gliclazide is long and varies between 12 and 20 hours.

Octreotide should be considered if hypoglycaemic despite the infusion of sufficient glucose. Octreotide is a somatostatin analogue and inhibits the secretion of many hormones, including insulin. It would be worth considering in this woman, due to her hypoglycaemia despite dextrose. IM glucagon may also be considered, particularly if IV access has proven difficult but its effects are dependent on available glycogen stores. Once euglycaemia is achieved, the patient ought to regain consciousness. Failure to do so ought to make one consider cerebral oedema.

It is too late to be considering activated charcoal as this is only considered 1-hour post-ingestion, and it is likely she ingested the sulfonylurea hours ago. Cyproheptadine is used in serotonin syndrome and carcinoid tumours. Dialysis is not indicated in sulphonylurea poisoning. Gliclazide would make things very much worse for this woman.

👍 👎 💬 Discuss (4) Improve

Next question

Hypoglycaemia ★

Causes

- insulinoma - increased ratio of proinsulin to insulin
- self-administration of insulin/sulphonylureas
- liver failure
- Addison's disease
- alcohol
  - causes exaggerated insulin secretion
  - mechanism is thought to be due to the effect of alcohol on the pancreatic microcirculation → redistribution of pancreatic blood flow from the exocrine into the endocrine parts → increased insulin secretion
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  - Confusion

    - Dizziness
- Severe and uncommon features of hypoglycaemia include:
    - Convulsion
    - Coma

Investigation of hypoglycaemia

- if the cause of hypoglycaemia is not clear then a combination of serum insulin and c-peptide levels can be measured
- remember that insulin and C-peptide are released in equimolar amounts from the pancreas, making C-peptide a marker of endogenous insulin production.

Insulin Level	**C-peptide Level**	**Interpretation**	**Potential Causes**
High	High	Endogenous insulin production	Insulinoma, Sulfonylurea use/abuse
High	Low	Exogenous insulin administration	Exogenous insulin overdose, Factitious disorder
Low	Low	Non-insulin-related cause	Alcohol-induced hypoglycaemia, Critical illness (e.g., sepsis), Adrenal insufficiency, Growth hormone deficiency, Fasting/starvation

Management of hypoglycaemia

- the following guidelines are based on the BNF hypoglycaemia treatment summary.
- in the community (for example, diabetes mellitus patients who inject insulin):
    - Initially, oral glucose 10-20g should be given in liquid, gel or tablet form
    - Alternatively, a propriety quick-acting carbohydrate may be given: GlucoGel or Dextrogel.

    - A 'HypoKit' may be prescribed which contains a syringe and vial of glucagon for IM or SC injection at home
- in a hospital setting:
    - If the patient is alert, a quick-acting carbohydrate may be given (as above)
    - If the patient is unconscious or unable to swallow, subcutaneous or intramuscular injection glucagon may be given.
    - Alternatively, intravenous 20% glucose solution may be given through a large vein

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **59.5%**

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Question 75 of 101

A 19-year-old man with no previous health issues presents to the emergency department with polyuria and extreme thirst.

His observations are as follows:

- Heart rate: 95/min
- Blood pressure: 100/63 mmHg
- Respiratory rate: 24/min
- Saturations: 94% on room air
- Temperature: 36.5°C

On examination, he is clinically dehydrated with dry mucous membranes and reduced skin turgor.

His initial venous blood gas is shown below:

pH	7.086	(7.350 - 7.450)
Na^+	141 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Bicarbonate	9 mmol/L	(22 - 29)
Glucose	31 mmol/L	(4-10)
Lactate	2.5 mmol/L	(< 2)

Finger-prick ketones are 6.0 mmol/L. You ask the nurse to prepare an insulin infusion.

What is the most appropriate initial fluid to give this patient?

○ 0.9% saline ✕

○ 0.9% saline with 40 mmol potassium ✕

○ 1.26% sodium bicarbonate ✕

○ 5% dextrose ×

○ 5% dextrose with 40 mmol potassium ×

Submit answer

Reference ranges

Score: **59.5%**

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← Question 75 of 101 ✗ ⚑ →

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Finger-prick ketones are 6.0 mmol/L. You ask the nurse to prepare an insulin infusion.

What is the most appropriate initial fluid to give this patient?

🟩	0.9% saline	65%
🟥	0.9% saline with 40 mmol potassium	33%
	1.26% sodium bicarbonate	1%

5% dextrose	0%
5% dextrose with 40 mmol potassium	0%

Diabetic ketoacidosis: isotonic saline should be used initially, even if the patient is severely acidotic

Important for me Less important

This patient is presenting with diabetic ketoacidosis (DKA) as evidenced by the elevated blood glucose, the elevated ketones, and the acidosis on his VBG. This is likely a first presentation of type 1 diabetes. This would explain his symptoms of polyuria and polydipsia, as well as his dehydration.

Treatment of DKA is with fixed rate insulin infusion, given at 0.1 units/kg/hour. The patient will also need significant quantities of intravenous fluids, given the large amount of fluids that are lost due to the diuretic effect of the high glucose levels. The preferred initial fluid is 0.9% saline, making **0.9% saline** the correct answer. Most guidelines advise giving this first litre of fluid over 1 hour. If the patient presents with shock (blood pressure < 90/60), then a bolus can be given first.

This patient will need IV potassium replacement. Their total body potassium levels will be depleted due to urinary losses, and this will be exacerbated by insulin therapy, which will shift potassium from plasma into cells. However, given that the first bag of fluid will be given rapidly over 1 hour, it would not be safe to include 40 mmol of potassium in this bag. Potassium is generally infused at a maximum rate of 10 mmol/hour peripherally (20 mmol/hour with central access). This makes **0.9% saline with 40 mmol potassium** incorrect. The potassium will need to be added in subsequent bags of fluid.

1.26% sodium bicarbonate is not routinely used in the management of DKA. Whilst it may sometimes be given in critical care settings, it should not be used as the initial fluid of choice prior to routine treatment with saline and insulin.

5% dextrose and **5% dextrose with 40 mmol potassium** are both incorrect as the patient is already hyperglycaemic due to DKA, and one of the aims of initial treatment is to bring the glucose down. If the glucose levels come down with treatment but ketones remain elevated, there may be a role for administering additional intravenous dextrose alongside the insulin infusion. However, this is not yet the case and would not be appropriate.

👍 👎 Discuss Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology
- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features
- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points - glucose > 13.8 mmol/l - pH < 7.30 - serum bicarbonate <18 mmol/l - anion gap > 10 - ketonaemia	Key points - glucose > 11 mmol/l or known diabetes mellitus - pH < 7.3 - bicarbonate < 15 mmol/l - ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic
  - please see an example fluid regime below.
- insulin
  - an intravenous infusion should be started at 0.1 unit/kg/hour
  - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
  - serum potassium is often high on admission despite total body potassium being low
  - this often falls quickly following treatment with insulin resulting in hypokalaemia
  - potassium may therefore need to be added to the replacement fluids
  - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	Volume
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	Potassium replacement in mmol/L of infusion solution
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and

- bicarbonate > 15.0mmol/L

Further points

- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:

- gastric stasis
- thromboembolism
- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

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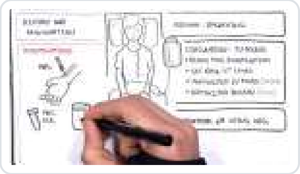
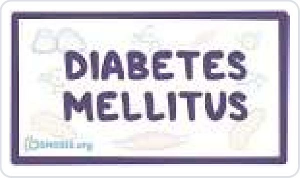
Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube

👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube

👍 9 👎 3

Report broken media

Score: **58.7%**

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Question 76 of 101 ✗

A 44-year-old woman is admitted to hospital complaining of a swollen breast for three days. She is otherwise well, having no medical problems. She is diagnosed by the surgical team with a breast abscess, which is drained and she is started on antibiotic treatment. Before being discharged, she is found to have elevated corrected calcium (2.79 mmol/L) and elevated parathyroid hormone (9.5 pmol/L).

She is reviewed by the endocrine team. She does not have any symptoms apart from those related to her breast abscess, and additional examination is unremarkable. Further tests are requested, showing that vitamin D levels are normal, 24-hour urine calcium is normal, and a DEXA scan is normal as well. She is advised to see her GP for annual blood tests for calcium levels and renal function.

She is diagnosed with primary hyperparathyroidism. What additional investigation should be used to monitor her?

	24-hour urine calcium annually	16%
	Breast ultrasound annually	5%
	Abdominal X-ray annually	9%
🟥	Abdominal ultrasound every three years	12%
🟩	DEXA scan every one to two years	59%

The correct answer is a DEXA scan. This patient has been incidentally found to have primary hyperparathyroidism and has no evidence of indications for parathyroidectomy. Monitoring should include renal function and DEXA scanning to identify any decline in renal function, worsening hypercalcaemia or osteoporosis. Any of these changes would be indications for surgery. Abdominal X-rays and ultrasound scanning may be useful in the acute setting to detect renal stones but are not recommended as monitoring. Urinary calcium useful at diagnosis to exclude hypocalciuric hypercalcaemia.

Source:

'Hypercalcaemia.' Clinical Knowledge Summaries. National Institute for Health and Care Excellence, Dec. 2014.

👍 👎 Discuss (8) Improve

Next question

Primary hyperparathyroidism ★

Primary hyperparathyroidism is caused by excess secretion of PTH resulting in hypercalcaemia. It is the most common cause of hypercalcaemia in outpatients and is often diagnosed following an incidental finding of an elevated serum calcium concentration. In 85% of cases a parathyroid adenoma is responsible.

Causes of primary hyperparathyroidism
- 85%: solitary adenoma
- 10%: hyperplasia
- 4%: multiple adenoma
- 1%: carcinoma

Around 80% of patients are asymptomatic and are diagnosed on routine blood tests. The symptomatic features of primary hyperparathyroidism may be remembered by the mnemonic: 'bones, stones, abdominal groans and psychic moans':
- polydipsia, polyuria
- depression
- anorexia, nausea, constipation
- peptic ulceration
- pancreatitis
- bone pain/fracture
- renal stones

- hypertension

Associations

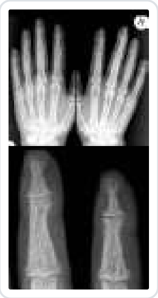
- hypertension
- multiple endocrine neoplasia: MEN I and II

Investigations

- bloods
    - raised calcium, low phosphate
    - PTH may be raised or (inappropriately, given the raised calcium) normal
- technetium-MIBI subtraction scan
- x-ray findings
    - pepperpot skull
    - osteitis fibrosa cystica

Treatment

- the definitive management is total parathyroidectomy
- conservative management may be offered if the calcium level is less than 0.25 mmol/L above the upper limit of normal AND the patient is > 50 years AND there is no evidence of end-organ damage
- patients not suitable for surgery may be treated with cinacalcet, a calcimimetic
    - a calcimimetic 'mimics' the action of calcium on tissues by allosteric activation of the calcium-sensing receptor



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

HYPERPARATHYROIDISM
SECONDARY

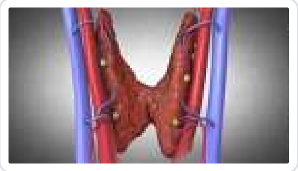
Understanding Hyperparathyroidism

Zero to Finals - YouTube 👍 13 👎 0

ENDOCRINOLOGY
HYPERPARATHYROIDISM

Hyperparathyroidism and the different types, causes, pathophysiology, treatment

Armando Hasudungan - YouTube 👍 2 👎 0



Parathyroid Glands and Hyperparathyroidism

Norman Parathyroid Center - YouTube 👍 2 👎 1

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Score: **57.9%**

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A 45-year-old lady is admitted to hospital with abdominal pain and malaise. She has no past medical history and takes no regular medications or supplements. Bloods tests show:

Ca 2+$^{+}$ 2.70 mmol/l
PO4 $^{+}$ 1.2 mmol/l
Creatinine 60 µmol/l

Chest X-ray - normal appearances

She denies taking any medications or supplements. Her chest X-ray is normal in appearance,and renal function normal. You ring the GP and find out her calcium was also slightly raised 8 years ago. What is the most likely diagnosis?

Secondary hyperparathyroidism	3%
Malignancy with bony metastasis	1%
Primary hyperparathyroidism	33%
[✓ Familial hypocalciuric hypercalcaemia]	61%
Sarcoidosis	2%

PO4 would normally be low in primary hyperparathyroidism. Her renal function is normal excluding secondary hyperparathyroidism. Sarcoidosis is unlikely with a normal CXR. This leaves malignancy or familial hypocalciuric hypercalcaemia. Although malignancy is possible her raised Ca2+ 8 years makes familial hypocalciuric hypercalcaemia more likely.

Discuss (5) Improve

Next question

Familial benign hypocalciuric hypercalcaemia ★

Familial benign hypocalciuric hypercalcaemia is a rare autosomal dominant disorder characterised by asymptomatic hypercalcaemia. It is due to a defect in the calcium-sensing receptor and a decreased sensitivity to increases in extracellular calcium.

The parathyroid hormone level is often not suppressed, as would be expected in all non-hyperparathyrodism related cases of hypercalcaemia. This is due to the decreased sensitivity to increases in extracellular calcium.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

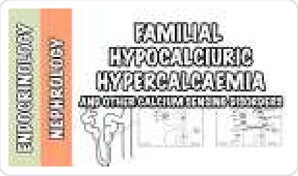
Links

Oxford Academic Website 👍 2 👎 0

Urine Calcium: Laboratory Measurement and Clinical Utility

Suggest link Report broken link

Media


ENDOCRINOLOGY
NEPHROLOGY
FAMILIAL HYPOCALCIURIC HYPERCALCAEMIA
AND OTHER CALCIUM SENSING DISORDERS

Familial Hypocalciuric Hypercalcemia + other calcium sensing disorders

Armando Hasudungan - YouTube 👍 7 👎 1

Report broken media

Score: **58.4%**

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← Question 78 of 101 →

A 55-year-old male presents with excessive sweating and headache.

You order an oral glucose tolerance test which is as follows:

Time (mins)	0	30	60	90	120
Growth hormone (ng/ml)	2.2	2.4	3.1	3.6	3.7
Glucose	9.1	10.8	11.5	12.8	15.2

Given the likely diagnosis, what other investigation will be required in the future?

- ○ Transesophageal echocardiography ×
- ○ Colonoscopy ×
- ○ Flexible sigmoidoscopy ×
- ○ CT colonography ×
- ○ No further investigations required ×

Submit answer

Dr. Jamil Alrajhi

Reference ranges ∨

Score: **58.4%**

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← Question 78 of 101 ✗ ⚐ →

A 55-year-old male presents with excessive sweating and headache.

You order an oral glucose tolerance test which is as follows:

Time (mins)	0	30	60	90	120
Growth hormone (ng/ml)	2.2	2.4	3.1	3.6	3.7
Glucose	9.1	10.8	11.5	12.8	15.2

Given the likely diagnosis, what other investigation will be required in the future?

	Transesophageal echocardiography	14%
🟩	Colonoscopy	55%
🟥	Flexible sigmoidoscopy	3%
	CT colonography	7%
	No further investigations required	22%

Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly

Important for me Less important

The clinical features and results of the oral glucose tolerance test confirm a diagnosis of acromegaly. In normal a rise in glucose should cause suppression of growth hormone (GH) to undetectable values.

Cardiovascular disease is highly prevalent in Acromegaly due to a combination of risk factors associated with this disease, in particular, secondary diabetes mellitus, hypertension and hyperlipidaemia. Patients should therefore be screened for cardiovascular risk factors and these should be managed aggressively. A transthoracic echo would be prudent to perform, however transesophageal echocardiography is unnecessary.

Patients with acromegaly have high levels of insulin like growth factor which predisposes to malignancy, in particularly colorectal carcinoma. Colonoscopy, flexible sigmoidoscopy and CT colonography can all be used to screen for colorectal carcinoma, however colonoscopy is the most sensitive test making this the best answer.

Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly.

👍 👎 Discuss (8) Improve

Next question

Acromegaly: management ★

Trans-sphenoidal surgery is the first-line treatment for acromegaly in the majority of patients.

If a pituitary tumour is inoperable or surgery unsuccessful then medication may be indicated:
- somatostatin analogue
  - directly inhibits the release of growth hormone
  - for example octreotide
  - effective in 50-70% of patients
- pegvisomant
  - GH receptor antagonist - prevents dimerization of the GH receptor
  - once daily s/c administration
  - very effective - decreases IGF-1 levels in 90% of patients to normal

    - doesn't reduce tumour volume therefore surgery still needed if mass effect
- dopamine agonists
    - for example bromocriptine
    - the first effective medical treatment for acromegaly, however now superseded by somatostatin analogues
    - effective only in a minority of patients

External irradiation is sometimes used for older patients or following failed surgical/medical treatment

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

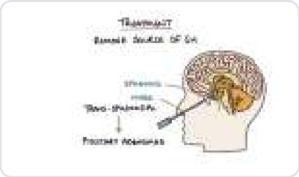
Postgraduate Medical Journal 👍 11 👎 4

Modern treatment of acromegaly

Suggest link

Report broken link

Media



Understanding Acromegaly

Zero To Finals - YouTube 👍 7 👎 1

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **57.7%**

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Question 79 of 101

A 54-year-old man presents to the diabetes clinic for review. He has had symptoms of polyuria, polydipsia and lethargy over the past few months, and his fasting glucose is elevated at 7.6 mmol/l. He has no history of diabetes in his family and is currently treated for hypertension and dyslipidaemia by his GP. On examination his blood pressure is 155/90 mmHg, pulse is 70 beats per minute and regular. His body mass index is 34 kg/m². Other blood tests of note include GAD+ antibodies, renal function is normal.

Which of the following is most appropriate with respect to managing his glucose control?

- ○ Gliclazide ×
- ○ Liraglutide ×
- ○ Metformin ×
- ○ Sitagliptin ×
- ○ Basal bolus insulin ×

Submit answer

Reference ranges

Score: **57.7%**

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Which of the following is most appropriate with respect to managing his glucose control?

🟥	Gliclazide	21%
	Liraglutide	7%
🟩	Metformin	47%
	Sitagliptin	2%
	Basal bolus insulin	23%

Up to 10% of patients who are thought to have type 2 diabetes are found to also be GAD autoantibody positive. These individuals are thought to progress faster to insulin start than patients with autoantibody negative type 2 diabetes, (within 3-5 years vs 7 years on average for 'standard' patients).

The management of these patients is the same as for patients with autoantibody negative disease, metformin as the initial therapy of choice. Weight reduction may delay progression to insulin, therefore insulin sparing strategies, at least during the first few years after diagnosis are the preferred intervention. These may include use of SGLT-2 inhibitors or GLP-1 agonists.

Diapedia reference:
http://www.diapedia.org/type-1-diabetes-mellitus/2104458121/lada-latent-autoimmune-diabetes-of-the-adult

👍 👎 Discuss (13) Improve

Next question

Diabetes mellitus: management of type 2 ★

NICE updated its guidance on the management of type 2 diabetes mellitus (T2DM) in 2022. This update reflected the advances in drug therapy and improved evidence regarding newer therapies such as SGLT-2 inhibitors.

> It's worthwhile thinking of the average patient who is taking metformin for T2DM, you can titrate up metformin and encourage lifestyle changes to aim for a HbA1c of 48 mmol/mol (6.5%), but should only add a second drug if the HbA1c rises to 58 mmol/mol (7.5%)

Dietary advice
- encourage high fibre, low glycaemic index sources of carbohydrates
- include low-fat dairy products and oily fish
- control the intake of foods containing saturated fats and trans fatty acids
- limited substitution of sucrose-containing foods for other carbohydrates is allowable, but care should be taken to avoid excess energy intake
- discourage the use of foods marketed specifically at people with diabetes
- initial target weight loss in an overweight person is 5-10%

HbA1c targets

This is an area which has changed in 2015

- individual targets should be agreed with patients to encourage motivation
- HbA1c should be checked every 3-6 months until stable, then 6 monthly
- NICE encourage us to consider relaxing targets on *'a case-by-case basis, with particular consideration for people who are older or frail, for adults with type 2 diabetes'*
- in 2015 the guidelines changed so HbA1c targets are now dependent on treatment:

Lifestyle or single-drug treatment

Management of T2DM	HbA1c target
Lifestyle	48 mmol/mol (6.5%)
Lifestyle + metformin	48 mmol/mol (6.5%)
Includes any drug which may cause hypoglycaemia (e.g. lifestyle + sulfonylurea)	53 mmol/mol (7.0%)

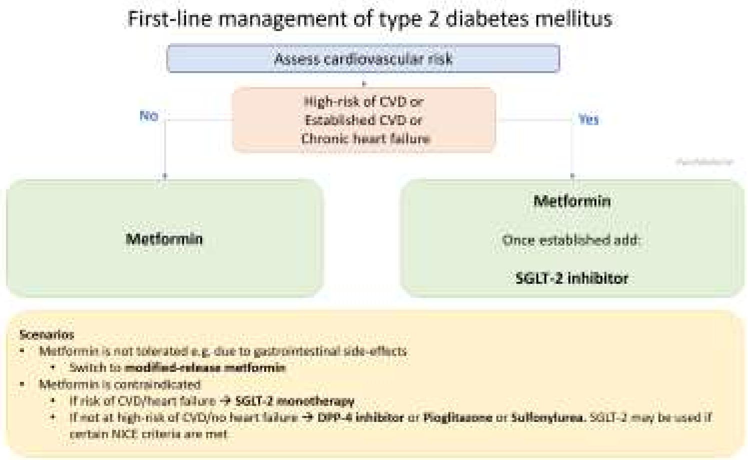
Practical examples

- a patient is newly diagnosed with HbA1c and wants to try lifestyle treatment first. You agree on a target of 48 mmol/mol (6.5%)
- you review a patient 6 months after starting metformin. His HbA1c is 51 mmol/mol (6.8%). You increase his metformin from 500mg bd to 500mg tds and reinforce lifestyle factors

Patient already on treatment

Management of T2DM	HbA1c target
Already on one drug, but HbA1c has risen to 58 mmol/mol (7.5%)	53 mmol/mol (7.0%)

Initial drug therapy


First-line management of type 2 diabetes mellitus
Assess cardiovascular risk
High-risk of CVD or
Established CVD or
Chronic heart failure
No
Yes
Metformin
Metformin
Once established add:
SGLT-2 inhibitor

Scenarios
- Metformin is not tolerated e.g. due to gastrointestinal side-effects
    - Switch to **modified-release metformin**
- Metformin is contraindicated
    - If risk of CVD/heart failure → **SGLT-2 monotherapy**
    - If not at high-risk of CVD/no heart failure → **DPP-4 inhibitor** or **Pioglitazone** or **Sulfonylurea.** SGLT-2 may be used if certain NICE criteria are met

Simplified initial management of type 2 diabetes mellitus

Metformin remains the first-line drug of choice in type 2 diabetes mellitus.

- metformin should be titrated up slowly to minimise the possibility of gastrointestinal upset
- if standard-release metformin is not tolerated then modified-release metformin should be trialled

SGLT-2 inhibitors

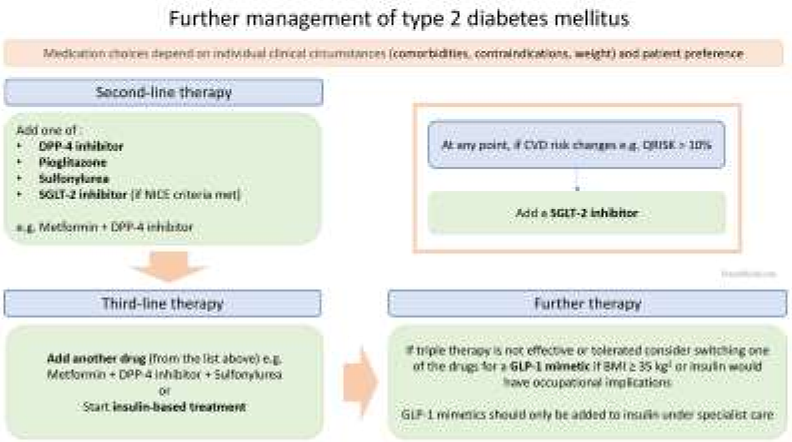
- should also be given in addition to metformin if any of the following apply:
    - the patient has a high risk of developing cardiovascular disease (CVD, e.g. QRISK ≥ 10%)
    - the patient has established CVD
    - the patient has chronic heart failure
- metformin should be established and titrated up before introducing the SGLT-2 inhibitor
- SGLT-2 inhibitors should also be started at any point if a patient develops CVD (e.g. is diagnosed with ischaemic heart disease), a

QRISK ≥ 10% or chronic heart failure

If metformin is contraindicated

- if the patient has a risk of CVD, established CVD or chronic heart failure:
    - SGLT-2 monotherapy
- if the patient doesn't have a risk of CVD, established CVD or chronic heart failure:
    - DPP-4 inhibitor or pioglitazone or a sulfonylurea
    - SGLT-2 may be used if certain NICE criteria are met

Further drug therapy if HbA1c targets are not met


Further management of type 2 diabetes mellitus
Medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference
Second-line therapy
Add one of :
DPP-4 inhibitor
Pioglitazone
Sulfonylurea
SGLT-2 inhibitor (if NICE criteria met)
e.g. Metformin + DPP-4 inhibitor
At any point, if CVD risk changes e.g. QRISK > 10%
Add a SGLT-2 inhibitor
Third-line therapy
Add another drug (from the list above) e.g. Metformin + DPP-4 inhibitor + Sulfonylurea
or
Start insulin-based treatment
Further therapy
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic if BMI ≥ 35 kg¹ or insulin would have occupational implications
GLP-1 mimetics should only be added to insulin under specialist care

Simplified further management of type 2 diabetes mellitus if HbA1c targets are not met

If the HbA1c has risen to 58 mmol/mol (7.5%) then further treatment is indicated. Given the large number of drug therapy options, medication choices depend on individual clinical circumstances (comorbidities, contraindications, weight) and patient preference.

Second-line therapy

Dual therapy - add one of the following:

- metformin + DPP-4 inhibitor
- metformin + pioglitazone

- metformin + sulfonylurea
- metformin + SGLT-2 inhibitor (if NICE criteria met)

Third-line therapy

If a patient does not achieve control on dual therapy then the following options are possible:
- metformin + DPP-4 inhibitor + sulfonylurea
- metformin + pioglitazone + sulfonylurea
- metformin + (pioglitazone or sulfonylurea or DPP-4 inhibitor) + SGLT-2 if certain NICE criteria are met
- insulin-based treatment

Further therapy

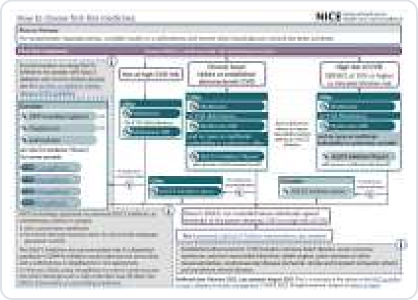
If triple therapy is not effective or tolerated consider switching one of the drugs for a GLP-1 mimetic:
- BMI ≥ 35 kg/m² and specific psychological or other medical problems associated with obesity or
- BMI < 35 kg/m² and for whom insulin therapy would have significant occupational implications or weight loss would benefit other significant obesity-related comorbidities
- only continue if there is a reduction of at least 11 mmol/mol [1.0%] in HbA1c and a weight loss of at least 3% of initial body weight in 6 months

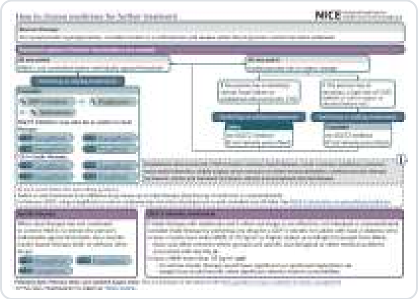
GLP-1 mimetics should only be added to insulin under specialist care

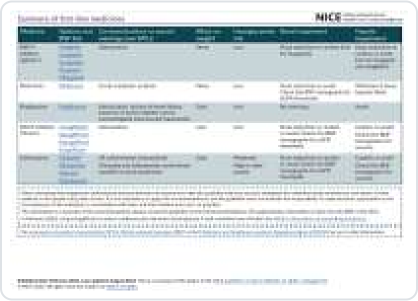
Starting insulin
- metformin should be continued. In terms of other drugs NICE advice: *'Review the continued need for other blood glucose-lowering therapies'*
- NICE recommend starting with human NPH insulin (isophane, intermediate-acting) taken at bed-time or twice daily according to need

Practical examples

- you review an established type 2 diabetic on maximum dose metformin. Her HbA1c is 55 mmol/mol (7.2%). You do not add another drug as she has not reached the threshold of 58 mmol/mol (7.5%)
- a type 2 diabetic is found to have an HbA1c of 62 mmol/mol (7.8%) at annual review. They are currently on maximum dose metformin. You elect to add a sulfonylurea







Risk factor modification

Hypertension

- **blood pressure targets are the same as for patients without type 2 diabetes** (see table below)
- ACE inhibitors or angiotensin II receptor blockers (ARB) are first-line
    - an ARB is preferred if the patient has a black African or African-Caribbean family origin

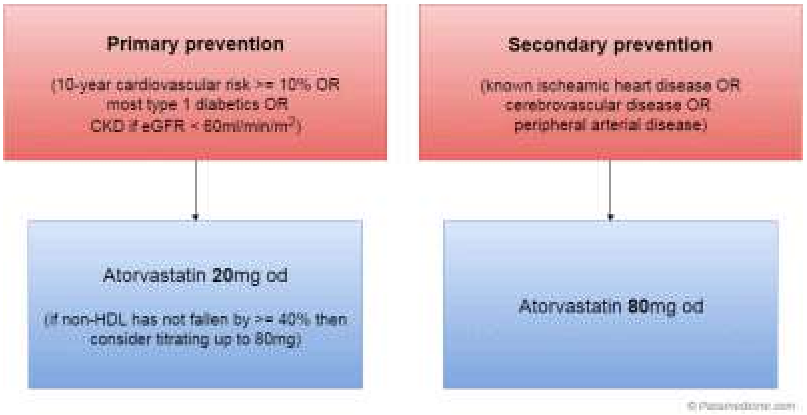
	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Antiplatelets

- should not be offered unless a patient has existing cardiovascular disease

Lipids

- following the 2014 NICE lipid modification guidelines only patients with a 10-year cardiovascular risk > 10% (using QRISK2) should be offered a statin. The first-line statin of choice is atorvastatin 20mg on


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin **20**mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin **80**mg od
© Passmedicine.com

Graphic showing choice of statin.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 33 👎 40

2014 Lipid modification guidelines

SIGN 👍 36 👎 41

2010 Management of diabetes

NICE 👍 56 👎 48

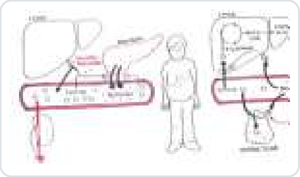
2022 Type 2 diabetes guidelines

7 minute medics 👍 44 👎 35

Basics of type 2 diabetes - podcast

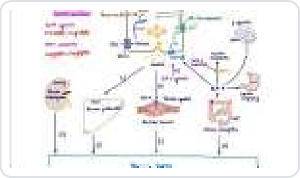
Suggest link Report broken link

Media



Diabetes Type II Pathophysiology

Armando Hasudungan - YouTube 👍 5 👎 0



Type 2 diabetes agents and their mechanism of action

Brandl's Basics - YouTube 👍 14 👎 4

Pharmacology of drugs used to treat type 2 diabetes

Brandl's Basics - YouTube 👍 5 👎 2

Report broken media

Score: **57%**

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Question 80 of 101

A 66-year-old man is referred to the endocrinology outpatient department with resistant hypertension and hypokalaemia. He is asymptomatic. He is on regular ramipril, amlodipine, indapamide and doxazosin. He has a further past medical history of hypercholesterolaemia. He has smoked five cigarettes daily for thirty years and drinks 2-3 bottles of wine per week. He is a non-executive director of a large multinational company.

His observations are heart rate 76 beats per minute, blood pressure 181/88 mmHg, respiratory rate 12/minute, oxygen saturations 97% on room air. He is apyrexial and appears clinically well. An examination is unremarkable other than a raised body mass index of 35.2 kg/m^2.

Blood tests:

Hb	141 g/L	Male: (135-180) Female: (115 - 160)
Platelets	222 * 10^9/L	(150 - 400)
WBC	6.2 * 10^9/L	(4.0 - 11.0)
Na$^+$	138 mmol/L	(135 - 145)
K$^+$	2.9 mmol/L	(3.5 - 5.0)
Urea	4.6 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Cortisol	589 nmol/L	(119 - 618)
Aldosterone: renin ratio	increased	

A CT of the abdomen demonstrates bilateral adrenal enlargement and adrenal vein sampling demonstrates the production of excess aldosterone bilaterally.

What is the most appropriate treatment?

- ○ Chemotherapy ×
- ○ IV hydrocortisone ×
- ○ Radiotherapy ×
- ○ Spironolactone ×
- ○ Surgery ×

Submit answer

Reference ranges ⌄

Score: **57%**

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Dr. Jamil Alrajhi

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CRP	3 mg/L	(< 5)
Cortisol	589 nmol/L	(119 - 618)
Aldosterone: renin ratio	increased	

A CT of the abdomen demonstrates bilateral adrenal enlargement and adrenal vein sampling demonstrates the production of excess aldosterone bilaterally.

What is the most appropriate treatment?

Chemotherapy	1%
IV hydrocortisone	2%
Radiotherapy	1%
Spironolactone	81%
Surgery	15%

Primary hyperaldosteronism: manage with spironolactone

Important for me Less important

Spironolactone is the correct answer. Primary hyperaldosteronism caused by bilateral adrenal hyperplasia is treated by a mineralocorticoid receptor antagonist such as spironolactone.

Chemotherapy is the wrong answer. The first-line treatment for bilateral adrenal hyperplasia causing primary hyperaldosteronism is a mineralocorticoid receptor antagonist such as spironolactone. If a patient had disseminated malignancy caused by an adrenal carcinoma, then this may be an option.

IV hydrocortisone is incorrect. This is the treatment for acute hypoadrenalism i.e. an Addisonian crisis. The patient's cortisol is within normal limits and he has no clinical features of hypoadrenalism.

Radiotherapy is incorrect. This type of treatment may be indicated for adrenal carcinoma. However, cancer is more likely to be a unilateral mass-like lesion, rather than bilateral diffuse enlargement.

Surgery is incorrect. This would be the correct option if the patient had a unilateral adrenal adenoma secreting excess aldosterone. This is not the case here.

Discuss (5) Improve

Next question

Primary hyperaldosteronism

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes

- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

Features

- **hypertension**
  - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
  - e.g. muscle weakness
  - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

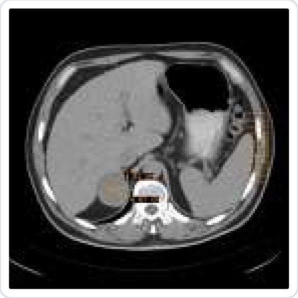
Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
  - hypertension with hypokalemia
  - treatment-resistant hypertension

- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
  - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
  - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

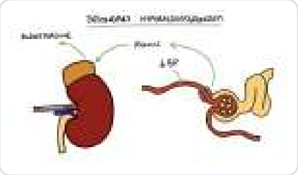
Suggest link Report broken link

Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

Report broken media

Score: **57.5%**

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A 42-year-old woman presents to the eye casualty with a 3-week history of gritty, painful eyes and photophobia. She was diagnosed with Graves' disease 6 months ago and is currently on carbimazole. On examination, she has bilateral proptosis, lid retraction, and conjunctival injection. Her visual acuity is 6/9 in both eyes. Slit lamp examination reveals punctate epithelial erosions on both corneas. She smokes 20 cigarettes per day.

What is the most appropriate next step in management?

- ○ Intravenous methylprednisolone ×
- ○ Orbital radiotherapy ×
- ○ Artificial tears and smoking cessation advice ×
- ○ Urgent orbital decompression surgery ×
- ○ Switch from carbimazole to propylthiouracil ×

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **57.5%**

1 ✓
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What is the most appropriate next step in management?

🟥	Intravenous methylprednisolone	46%
	Orbital radiotherapy	1%
🟩	Artificial tears and smoking cessation advice	39%
	Urgent orbital decompression surgery	10%
	Switch from carbimazole to propylthiouracil	3%

Exposure keratopathy is the most common complication of thyroid eye disease

Important for me Less important

This patient is presenting with symptoms and signs consistent with exposure keratopathy secondary to thyroid eye disease (TED). The gritty sensation, pain, photophobia, and punctate epithelial erosions on the cornea are classic features of exposure keratopathy, which is the most common complication of TED. The patient has proptosis and lid retraction, which lead to inadequate corneal coverage and disruption of the tear film, resulting in corneal dryness and epithelial damage.

Artificial tears and smoking cessation advice is the most appropriate initial management for this patient. Artificial tears will provide lubrication to the exposed cornea, reducing symptoms and preventing further epithelial damage. Smoking cessation is crucial as smoking is the most important modifiable risk factor for TED progression. Studies show that smokers have more severe disease, poorer response to treatment, and higher recurrence rates. The patient's current moderate disease (with preserved visual acuity of 6/9) warrants conservative management before considering more invasive options. Regular lubrication and eliminating smoking can significantly improve symptoms and may prevent disease progression.

Intravenous methylprednisolone is used for moderate to severe active TED, particularly when there is sight-threatening disease such as optic neuropathy or severe corneal exposure. While this patient has active TED, her condition is not yet severe enough to warrant systemic steroid therapy with its significant side effects. The absence of visual acuity reduction below 6/9 suggests that optic nerve compression is not present, making high-dose steroids inappropriate as first-line treatment.

Orbital radiotherapy is typically reserved for active, moderate to severe TED, particularly for patients with diplopia and restricted eye movements. It works by targeting the inflammatory cells in the orbit. This patient does not have documented extraocular muscle involvement or diplopia, and radiotherapy would not address the immediate issue of corneal exposure. Additionally, radiotherapy is usually considered after failure of steroid therapy or as an adjunct to steroids.

Urgent orbital decompression surgery is indicated for sight-threatening optic neuropathy or severe corneal exposure that is unresponsive to medical therapy. This patient's condition, while uncomfortable, is not sight-threatening at present. Her visual acuity is preserved at 6/9, and the corneal changes are at the punctate erosion stage rather than ulceration or perforation. Surgical intervention would be excessive at this stage of disease.

Switching from carbimazole to propylthiouracil would not address the ocular manifestations of TED. While managing the underlying thyroid dysfunction is important in TED, changing the type of antithyroid medication has not been shown to significantly impact the course of the eye disease. The activity of TED often runs independently of thyroid function, and many patients develop eye disease while euthyroid.

👍 👎 Discuss (1) Improve

Next question

Thyroid eye disease ★

Thyroid eye disease affects between 25-50% of patients with Graves' disease.

Pathophysiology

- it is thought to be caused by an autoimmune response against an autoantigen, possibly the TSH receptor → retro-orbital inflammation
- the inflammation results in glycosaminoglycan and collagen deposition in the muscles

Prevention

- smoking is the most important modifiable risk factor for the development of thyroid eye disease
- radioiodine treatment may increase the inflammatory symptoms seen in thyroid eye disease. In a recent study of patients with Graves' disease around 15% developed, or had worsening of, eye disease. Prednisolone may help reduce the risk

Features

- the patient may be eu-, hypo- or hyperthyroid at the time of presentation
- exophthalmos
- conjunctival oedema
- optic disc swelling
- ophthalmoplegia
- inability to close the eyelids may lead to sore, dry eyes. If severe and untreated patients can be at risk of exposure keratopathy

Management

- smoking cessation

- topical lubricants may be needed to help prevent corneal inflammation caused by exposure
- steroids
- radiotherapy
- surgery

Complications

Exposure keratopathy
- this is the most common complication of thyroid eye disease
- due to eyelid retraction and proptosis (exophthalmos) → cornea becomes excessively exposed, disrupting the normal tear film → dryness, irritation, and corneal ulceration
- symptoms include foreign body sensation, pain, and photophobia
- in severe cases, it can lead to corneal scarring and vision impairment.

Optic neuropathy
- one of the most serious complications of thyroid eye disease
- occurs when enlarged extraocular muscles compress the optic nerve at the apex of the orbit → a reduction in visual acuity, colour vision deficits, and visual field defect
- it requires urgent medical intervention to prevent permanent vision loss.

Strabismus and diplopia
- fibrosis and enlargement of the extraocular muscles can result in restrictive strabismus → misalignment of the eyes → double vision (diplopia)
- this not only affects visual function but can also significantly impair the quality of life.

Monitoring patients with established thyroid eye disease

For patients with established thyroid eye disease the following symptoms/signs should indicate the need for urgent review by an ophthalmologist (see EUGOGO guidelines):

- unexplained deterioration in vision
- awareness of change in intensity or quality of colour vision in one or both eyes
- history of eye suddenly 'popping out' (globe subluxation)
- obvious corneal opacity
- cornea still visible when the eyelids are closed
- disc swelling

+ Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Consensus Statement of the European Group on Graves' Orbitopathy 👍 9 👎 8

Management of Graves' Orbitopathy

Suggest link

Report broken link

Score: **56.8%**

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Question 82 of 101

A 32-year-old man attends as a day case for minor surgery. You are asked to see him on arrival as his blood pressure is 197/135mmHg. You organise ambulatory blood pressure monitoring which confirms an average BP of 184/111mmHg. Blood tests are sent and the results are below.

Na^+	144 mmol/L	(135 - 145)
K^+	2.4 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	71 µmol/L	(55 - 120)

What is the most appropriate initial treatment?

- ○ Amlodipine ×
- ○ Fludrocortisone ×
- ○ Oral potassium supplementation ×
- ○ Ramipril ×
- ○ Spironolactone ×

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **56.8%**

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Back to top

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Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	71 µmol/L	(55 - 120)

What is the most appropriate initial treatment?

Amlodipine	4%
Fludrocortisone	3%
Oral potassium supplementation	8%
Ramipril	13%
Spironolactone	72%

Primary hyperaldosteronism: manage with spironolactone

Important for me Less important

The correct answer is **spironolactone**. Such marked hypertension in a young patient should prompt consideration of an underlying cause and the hypokalaemia would be very suggestive of primary hyperaldosteronism. This is managed initially with mineralocorticoid receptor antagonists such as spironolactone. These are associated with improvements in both his hypokalaemia and hypertension.

Amlodipine is incorrect. This is a dihydropyridine calcium channel antagonist usually used in the management of essential hypertension. It is unlikely to be effective in primary hyperaldosteronism. Other calcium channel antagonists such as verapamil may help control hypertension while awaiting the results of confirmatory tests, but amlodipine is not recommended.

Fludrocortisone is incorrect. This is a mineralocorticoid with more of a use in adrenal insufficiency. It is likely to worsen hypertension here. It can occasionally be used in a suppression test to confirm a diagnosis of primary hyperaldosteronism but has no place in treatment.

Oral potassium supplementation is incorrect. While this may lead to a temporary improvement in the patient's hypokalaemia, this is unlikely to be sustained. It will also not treat his hypertension. Such treatments may be used in other causes of hypokalaemia such as Bartter's or Gitelman's syndrome. However, these are generally associated with normotension or even hypotension.

Ramipril is incorrect. This would be an appropriate treatment for essential hypertension. However, this patient's young age should raise suspicion of a secondary cause and the hypokalaemia points to primary hyperaldosteronism as the most likely. Ramipril is unlikely to improve this patient's blood pressure significantly and can also interfere with the diagnostics in this condition.

👍 👎 💬 Discuss (6) Improve

Next question

Primary hyperaldosteronism ★

Primary hyperaldosteronism was previously thought to be most commonly caused by an adrenal adenoma, termed Conn's syndrome. However, recent studies have shown that bilateral idiopathic adrenal hyperplasia is the most common cause. Differentiating between the two is important as this determines treatment.

Causes

- bilateral idiopathic adrenal hyperplasia: the cause of around 60-70% of cases
- adrenal adenoma: 20-30% of cases
- unilateral hyperplasia
- familial hyperaldosteronism
- adrenal carcinoma

Features

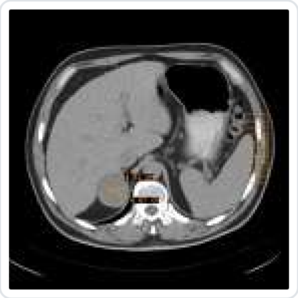
- **hypertension**
  - increasingly recognised but still underdiagnosed cause of hypertension
- **hypokalaemia**
  - e.g. muscle weakness
  - this is a classical feature in exams but studies suggest this is seen in only 10-40% of patients, and is more common with adrenal adenomas
- metabolic alkalosis

Investigations

- guidelines vary but certain patients should be screened for primary hyperaldosteronism, e.g.
  - hypertension with hypokalemia
  - treatment-resistant hypertension
- the 2016 Endocrine Society recommend that a plasma **aldosterone/renin ratio is the first-line investigation** in suspected primary hyperaldosteronism
  - should show high aldosterone levels alongside low renin levels (negative feedback due to sodium retention from aldosterone)
- following this a high-resolution CT abdomen and adrenal vein sampling is used to differentiate between unilateral and bilateral sources of aldosterone excess
  - if the CT is normal adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia

Management

- adrenal adenoma: surgery (laparoscopic adrenalectomy)
- bilateral adrenocortical hyperplasia: aldosterone antagonist e.g. spironolactone



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Next question

Textbooks

High-yield textbook

Extended textbook

Links

The Endocrine Society 👍 16 👎 13

2016: The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment

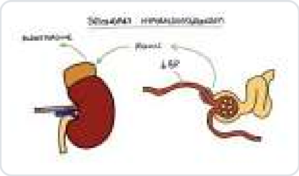
Suggest link Report broken link

Media

HYPERALDOSTERONISM

Hyperaldosteronism - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 5 👎 0



Hyperaldosteronism and Conn's Syndrome

Zero To Finals - YouTube 👍 14 👎 1

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Score: **57.3%**

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← Question 83 of 101 →

A 32-year-old female presents to the infertility clinic with an inability to conceive. She is overweight, with a body-mass index of 32 kg/m², and has noticed increased hair growth over her face and chest over the last 12 months. Her periods are irregular and she has also noticed a deepening of her voice. An ultrasound of the pelvis has revealed the presence of multiple cysts in both ovaries. She has been treated with cyproterone acetate for her hirsutism but was informed that she should not attempt conception whilst on the drug. She now wishes to conceive.

On examination, she has a cushingoid appearance, with abdominal striae and her blood pressure is 140/85 mmHg.

Laboratory investigations reveal:

9:00 am Cortisol	710 nmol/l (170 700 nmol/l)
LH	28 iU/l (1 20 iU/l)
Basal FSH	4.7 iU/l (1.0 8.8 iU/l)
DHEAS	509 µg/dl (31 228 µg/dl)
Prolactin	602 mU/l (<360 mU/l)
17 OH Progesterone	54 ng/dl (<80 ng/dl)

Which of the following treatment options would be most appropriate for the treatment of infertility?

- ○ Metformin ✕
- ○ Spironolactone ✕
- ○ Reverse circadian rhythm steroids ✕
- ○ Clomiphene citrate ✕

○ Cabergoline ×

Submit answer

Reference ranges

Dr. Jamil Alrajhi

Score: **57.3%**

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← Question 83 of 101 ✗ ⚐ →

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Prolactin	602 mU/l (<360 mU/l)
17 OH Progesterone	54 ng/dl (<80 ng/dl)

Which of the following treatment options would be most appropriate for the treatment of infertility?

Metformin	13%
Spironolactone	2%
🟥 Reverse circadian rhythm steroids	7%
🟩 Clomiphene citrate	69%

Cabergoline 8%

The Rotterdam criteria for the diagnosis of PCOS requires at least two of the following

- Clinical or biochemical evidence of hyperandrogenism.
- Evidence of oligo- or anovulation.
- Presence of polycystic ovaries on ultrasound.

Multiple clinical trials have been conducted to assess which drug is the most appropriate in aiding fertility. An article published in the New England journal of Medicine entitled Clomiphene, Metformin, or Both for Infertility in the Polycystic Ovary Syndrome concluded that Clomiphene is superior to metformin in achieving live birth in infertile women with the polycystic ovary syndrome, although multiple birth is a complication(N Engl J Med 2007; 356:551-566 February 8, 2007).

Another article Status of clomiphene citrate and metformin for infertility in PCOS (Trends Endocrinol Metab. 2012 Oct;23(10):533-43) published the following results:
'Though widely used, there is uncertainty about the effectiveness and adverse effects of metformin and clomiphene citrate (CC) for infertility in polycystic ovary syndrome (PCOS). A systematic review (SR) of the best available evidence suggests that both CC and metformin are better than placebo for increasing ovulation and pregnancy rates, but CC is more effective than metformin for ovulation, pregnancy and live-birth rates, in PCOS patients with body mass index (BMI) >30.'

In PCOS, serum prolactin may also be marginally raised, but the levels seldom exceed 1500 mU/l.

Reverse circadian rhythm steroids are used in the treatment of congenital adrenal hyperplasia, whilst cabergoline is used for the medical management of hyperprolactinemia.

Spironolactone has antiandrogenic activity and can cause improvements in hirsutism in PCOS but has no bearing on fertility.

👍 👎 Discuss (7) Improve

Next question

Polycystic ovarian syndrome: management

Polycystic ovarian syndrome (PCOS) is a complex condition of ovarian dysfunction thought to affect between 5-20% of women of reproductive age. Management is complicated and problem based partly because the aetiology of PCOS is not fully understood. Both hyperinsulinaemia and high levels of luteinizing hormone are seen in PCOS and there appears to be some overlap with the metabolic syndrome.

General
- weight reduction if appropriate
- if a women requires contraception then a combined oral contraceptive (COC) pill may help regulate her cycle and induce a monthly bleed (see below)

Hirsutism and acne
- a COC pill may be used help manage hirsutism. Possible options include a third generation COC which has fewer androgenic effects or co-cyprindiol which has an anti-androgen action. Both of these types of COC may carry an increased risk of venous thromboembolism
- if doesn't respond to COC then topical eflornithine may be tried
- spironolactone, flutamide and finasteride may be used under specialist supervision

Infertility
- weight reduction if appropriate
- the management of infertility in patients with PCOS should be supervised by a specialist. There is an ongoing debate as to whether metformin, clomifene or a combination should be used to stimulate ovulation

- a 2007 trial published in the New England Journal of Medicine suggested clomifene was the most effective treatment. There is a potential risk of multiple pregnancies with anti-oestrogen* therapies such as clomifene. The RCOG published an opinion paper in 2008 and concluded that on current evidence metformin is not a first line treatment of choice in the management of PCOS
- metformin is also used, either combined with clomifene or alone, particularly in patients who are obese
- gonadotrophins

*work by occupying hypothalamic oestrogen receptors without activating them. This interferes with the binding of oestradiol and thus prevents negative feedback inhibition of FSH secretion

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

ESHRE (European Society of Human Reproduction and Embryology) 👍 3 👎 5

[2018 International Evidence Based Guideline for the Assessment and Management of PCOS]

Clinical Knowledge Summaries 👍 4 👎 6

Polycystic ovarian syndrome

Royal College of Obstetricians and Gynaecologists 👍 8 👎 4

2014 Long-term Consequences of Polycystic Ovary Syndrome

RCOG 👍 10 👎 4

Opinion paper on metformin in PCOS

Patient.info 👍 5 👎 2

Polycystic ovarian syndrome review

Suggest link Report broken link

Score: **56.6%**

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Back to top

A 56-year-old man with type 1 diabetes arrives to hospital with one day of diarrhoea and vomiting. He felt well this morning but suffered indigestion and took some antacids without relief. The indigestion which he describes as a burning pain in the chest spreading into his throat lasted 4 hours however his lunch time blood glucose continued to rise despite several insulin boluses. He has been otherwise well over the past week and denies skipping meals, changes in exercise or any missed doses of his insulin. He smokes twenty cigarettes a day and maintains his diabetes on a basal bolus regime with correction doses based on carbohydrate counting.

On examination, he is sweaty with dry mucosa. His heart rate is 125/min, respiratory rate is 28/min with prolonged expiration phases, blood pressure is 110/90mmHg and temperature is 37ºC.

The nurse does his blood glucose which is 27mmol/l and his blood ketones are 4mmol/l.

Hb	130 g/l	Na^+	133 mmol/l
Platelets	356 * 10^9/l	K^+	4.5 mmol/l
WBC	9.8 * 10^9/l	Urea	6.2 mmol/l
Neuts	7.5 * 10^9/l	Creatinine	98 µmol/l
Lymphs	1.0 * 10^9/l	CRP	34 mg/l
Eosin	0.1 * 10^9/l		
HbA1c 48mmol/mol (normal range <42)			

ECG	deep T-wave inversion in V1-V4
Chest x-ray	nil acute seen

What is the likeliest cause that precipitated this presentation?

○ Missed insulin dose ×

○ Faulty blood glucose monitor ×

○ Lower respiratory tract infection ×

○ Myocardial infarction ×

○ Gastritis ×

Submit answer

Reference ranges ⌄

Score: **56.6%**

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Eosin	0.1 * 10^9/l		
HbA1c 48mmol/mol (normal range <42)			

ECG	deep T-wave inversion in V1-V4
Chest x-ray	nil acute seen

What is the likeliest cause that precipitated this presentation?

Missed insulin dose	5%
Faulty blood glucose monitor	2%
Lower respiratory tract infection	4%
Myocardial infarction	80%
Gastritis	9%

Myocardial infarction can precipitate diabetic ketoacidosis

Important for me Less important

This patient is a well controlled type 1 diabetic presenting with diabetic ketoacidosis (DKA) with no clear precipitant. Given a normal HbA1c and denial of any lifestyle changes, we should assume that he has not just missed his insulin. The blood glucose monitors are very reliable these days and rarely are wrong. Gastritis would not cause DKA. Myocardial infarction (MI) and lower respiratory tract infections can both precipitate DKA and given the burning chest pain and ECG changes, it would be warranted to do a troponin immediately as an MI this is the likeliest cause of DKA. To remember the causes of DKA, think 3 'I's which are insulin (missed), infection or infarction (i.e. MI).

👍 👎 Discuss (5) Improve

Next question

Diabetic ketoacidosis ★

Diabetic ketoacidosis (DKA) may be a complication of existing type 1 diabetes mellitus or be the first presentation, accounting for around 6% of cases. Rarely, under conditions of extreme stress, patients with type 2 diabetes mellitus may also develop DKA.

Whilst DKA remains a serious condition, mortality rates have decreased from 8% to under 1% in the past 20 years.

Pathophysiology

- DKA is caused by uncontrolled lipolysis (not proteolysis) which results in an excess of free fatty acids that are ultimately converted to ketone bodies

The most common precipitating factors of DKA are infection, missed insulin doses and myocardial infarction.

Features

- abdominal pain
- polyuria, polydipsia, dehydration
- Kussmaul respiration (deep hyperventilation)
- Acetone-smelling breath ('pear drops' smell)

Diagnostic criteria

American Diabetes Association (2009)	**Joint British Diabetes Societies (2013)**
Key points • glucose > 13.8 mmol/l • pH < 7.30 • serum bicarbonate <18 mmol/l • anion gap > 10 • ketonaemia	Key points • glucose > 11 mmol/l or known diabetes mellitus • pH < 7.3 • bicarbonate < 15 mmol/l • ketones > 3 mmol/l or urine ketones ++ on dipstick

Management

Main principles of management

- fluid replacement
  - most patients with DKA are deplete around 5-8 litres
  - isotonic saline is used initially, even if the patient is severely acidotic

    - please see an example fluid regime below.
- insulin
    - an intravenous infusion should be started at 0.1 unit/kg/hour
    - once blood glucose is < 14 mmol/l an infusion of 10% dextrose should be started at 125 mls/hr *in addition* to the 0.9% sodium chloride regime
- correction of electrolyte disturbance
    - serum potassium is often high on admission despite total body potassium being low
    - this often falls quickly following treatment with insulin resulting in hypokalaemia
    - potassium may therefore need to be added to the replacement fluids
    - if the rate of potassium infusion is greater than 20 mmol/hour then cardiac monitoring may be required
- long-acting insulin should be continued, short-acting insulin should be stopped

JBDS example of fluid replacement regime for patient with a systolic BP on admission 90mmHg and over

Fluid	**Volume**
0.9% sodium chloride 1L	1000ml over 1st hour
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 2 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 4 hours
0.9% sodium chloride 1L with potassium chloride	1000ml over next 6 hours

Please note that slower infusion may be indicated in young adults (aged 18-25 years) as they are at greater risk of cerebral oedema.

JBDS potassium guidelines

Potassium level in first 24 hours (mmol/L)	**Potassium replacement in mmol/L of infusion solution**
Over 5.5	Nil
3.5-5.5	40
Below 3.5	Senior review as additional potassium needs to be given

Resolution

DKA resolution is defined as:
- pH >7.3 and
- blood ketones < 0.6 mmol/L and
- bicarbonate > 15.0mmol/L

Further points
- both the ketonaemia and acidosis should have been resolved within 24 hours. If this hasn't happened the patient requires senior review from an endocrinologist
- if the above criteria are met and the patient is eating and drinking switch to subcutaneous insulin
- the patient should be reviewed by the diabetes specialist nurse prior to discharge

Complications

Complications may occur from DKA itself or the treatment:
- gastric stasis
- thromboembolism

- arrhythmias secondary to hyperkalaemia/iatrogenic hypokalaemia
- iatrogenic due to incorrect fluid therapy: cerebral oedema*, hypokalaemia, hypoglycaemia
- acute respiratory distress syndrome
- acute kidney injury

* children/young adults are particularly vulnerable to cerebral oedema following fluid resuscitation in DKA and often need 1:1 nursing to monitor neuro-observations, headache, irritability, visual disturbance, focal neurology etc. It usually occurs 4-12 hours following commencement of treatment but can present at any time. If there is any suspicion a CT head and senior review should be sought

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

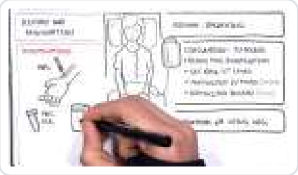
Joint British Inpatient Diabetes Societies Inpatient Care Group
The Management of Diabetic ketoacidosis in adults

👍 33 👎 23

Suggest link

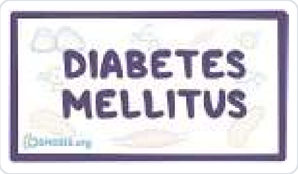
Report broken link

Media



Diabetic Ketoacidosis (Diabetes Type I) Management Summary

Armando Hasudungan - YouTube 👍 6 👎 2



Diabetes mellitus (type 1, type 2) & diabetic ketoacidosis (DKA) - causes & symptoms

Osmosis - YouTube 👍 9 👎 3

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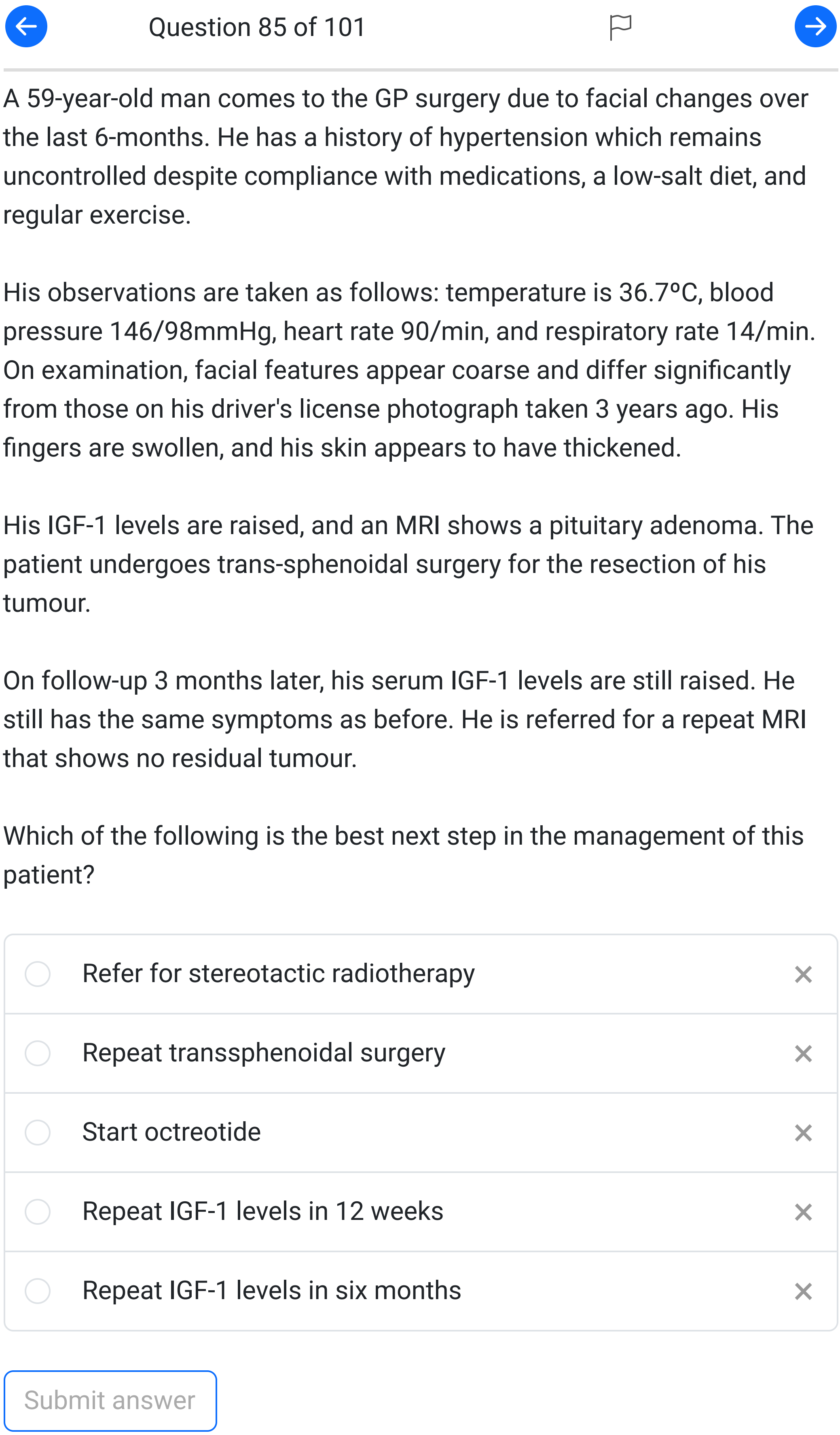

A 59-year-old man comes to the GP surgery due to facial changes over the last 6-months. He has a history of hypertension which remains uncontrolled despite compliance with medications, a low-salt diet, and regular exercise.

His observations are taken as follows: temperature is 36.7°C, blood pressure 146/98mmHg, heart rate 90/min, and respiratory rate 14/min. On examination, facial features appear coarse and differ significantly from those on his driver's license photograph taken 3 years ago. His fingers are swollen, and his skin appears to have thickened.

His IGF-1 levels are raised, and an MRI shows a pituitary adenoma. The patient undergoes trans-sphenoidal surgery for the resection of his tumour.

On follow-up 3 months later, his serum IGF-1 levels are still raised. He still has the same symptoms as before. He is referred for a repeat MRI that shows no residual tumour.

Which of the following is the best next step in the management of this patient?

- ○ Refer for stereotactic radiotherapy ×
- ○ Repeat transsphenoidal surgery ×
- ○ Start octreotide ×
- ○ Repeat IGF-1 levels in 12 weeks ×
- ○ Repeat IGF-1 levels in six months ×

Dr. Jamil Alrajhi

Reference ranges ⌄

Score: **57.1%**

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Which of the following is the best next step in the management of this patient?

	Refer for stereotactic radiotherapy	9%
🟥	Repeat transsphenoidal surgery	1%
🟩	Start octreotide	75%
	Repeat IGF-1 levels in 12 weeks	10%
	Repeat IGF-1 levels in six months	4%

Acromegaly: if patients are not suitable for trans-sphenoidal surgery, or have residual symptoms, then octreotide may be used

Important for me Less important

This patient's facial changes over time, coarse facial features on examination and elevated IGF-1 levels point towards a diagnosis of acromegaly. Since his MRI showed a pituitary adenoma, he was treated with transsphenoidal surgery. However, he remains symptomatic, and his IGF-1 levels have not reduced even after 3 months. Patients with residual symptoms need to repeat transsphenoidal surgery or start medical therapy with octreotide.

Since this patient has no residual tumour on his repeat MRI, he is not a candidate for repeat transsphenoidal surgery. Therefore, the most appropriate step for this patient is to start medical therapy with octreotide. Octreotide is a somatostatin analogue. It tends to decrease the size of the adenoma and its secretion of growth hormone.

Stereotactic radiotherapy is recommended for patients who fail medical therapy and is therefore not the best next step in the management of this patient.

Repeating IGF-1 levels again in 12 weeks is inappropriate as the IGF-1 level should have normalised at this follow-up 12 weeks after the transsphenoidal surgery. His repeat IGF-1 levels at 12 weeks are raised. He requires further medical or surgical management instead of follow-up in 12 weeks.

Patients with normal IGF-1 levels on a 12-week follow-up should have repeat IGF-1 levels at six months and then, annually.

👍 👎 Discuss (3) Improve

Next question

Acromegaly: management ★

Trans-sphenoidal surgery is the first-line treatment for acromegaly in the majority of patients.

If a pituitary tumour is inoperable or surgery unsuccessful then medication may be indicated:

- somatostatin analogue
    - directly inhibits the release of growth hormone
    - for example octreotide
    - effective in 50-70% of patients
- pegvisomant
    - GH receptor antagonist - prevents dimerization of the GH receptor
    - once daily s/c administration
    - very effective - decreases IGF-1 levels in 90% of patients to normal
    - doesn't reduce tumour volume therefore surgery still needed if mass effect
- dopamine agonists
    - for example bromocriptine
    - the first effective medical treatment for acromegaly, however now superseded by somatostatin analogues
    - effective only in a minority of patients

External irradiation is sometimes used for older patients or following failed surgical/medical treatment

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

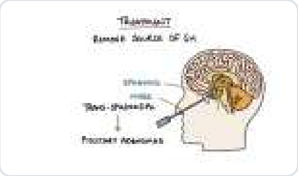
Links

Postgraduate Medical Journal 👍 11 👎 4

Modern treatment of acromegaly

Suggest link Report broken link

Media



Understanding Acromegaly

Zero To Finals - YouTube 👍 7 👎 1

ACROMEGALY

Acromegaly

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **56.5%**

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A 35-year-old woman presents to the emergency department with 3 days of fevers and sweating. She has a past medical history of Graves' disease and is not compliant with medication treatment. She smokes ten cigarettes daily and works in advertising.

Her observations are heart rate 146 beats per minute, blood pressure 154/99 mmHg, respiratory rate 24/minute, oxygen saturations 97% on room air and temperature 38.4ºC.

On examination, she is diaphoretic, tremulous and confused (Glasgow coma scale 14/15). Proptosis and chemosis are noted on examination of her eyes. There are bilateral crackles on chest auscultation and her JVP is elevated.

Blood tests:

Hb	124 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	5.3 * 10^9/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	26 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	99 u/L	(3 - 40)
Î³GT	74 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)
TSH	0.0 mIU/L	(0.2 - 5.5)

Free T4	81 pmol/L	(10 - 24.5)

Based on the likely diagnosis, what treatment is most appropriate from the listed options?

- ○ Antibiotics ×
- ○ Carbimazole ×
- ○ Propylthiouracil ×
- ○ Radioactive iodine therapy ×
- ○ Surgery ×

Submit answer

Reference ranges

Score: **56.5%**

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A 35-year-old woman presents to the emergency department with 3 days of fevers and sweating. She has a past medical history of Graves' disease and is not compliant with medication treatment. She smokes ten cigarettes daily and works in advertising.

Her observations are heart rate 146 beats per minute, blood pressure 154/99 mmHg, respiratory rate 24/minute, oxygen saturations 97% on room air and temperature 38.4ºC.

On examination, she is diaphoretic, tremulous and confused (Glasgow coma scale 14/15). Proptosis and chemosis are noted on examination of her eyes. There are bilateral crackles on chest auscultation and her JVP is elevated.

Blood tests:

Hb	124 g/L	Male: (135-180) Female: (115 - 160)
Platelets	$189 * 10^9$/L	(150 - 400)
WBC	$5.3 * 10^9$/L	(4.0 - 11.0)
Na^+	131 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	26 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	99 u/L	(3 - 40)
Î³GT	74 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)
TSH	0.0 mIU/L	(0.2 - 5.5)

Free T4	81 pmol/L	(10 - 24.5)

Based on the likely diagnosis, what treatment is most appropriate from the listed options?

Antibiotics	1%
Carbimazole	28%
Propylthiouracil	58%
Radioactive iodine therapy	8%
Surgery	5%

In thyroid storm, treat acutely with propylthiouracil rather than carbimazole or surgery

Important for me Less important

Propylthiouracil is the correct answer. The patient presents with ophthalmological features of Graves' disease in association with fever, confusion, tachycardia, tremor, hypertension and clinical evidence of heart failure. In the setting of significant hyperthyroidism this constitutes a 'thyroid storm'. Propylthiouracil has traditionally been the preferred anti-thyroid drug to be used in this setting due to its more rapid onset of action when compared with carbimazole and its additional ability to inhibit peripheral conversion of T4 to T3.

Antibiotics is not the right answer. Although this patient has a fever, she has a normal CRP and WCC and infection is less likely than thyroid storm given the clinical context.

Carbimazole is incorrect. This can be used in thyroid storm but is less preferred due to its slower onset of action.

Radioactive iodine therapy is incorrect. This can be used to manage Graves' disease but is not an option acutely for treating thyroid storm.

Surgery is incorrect. This is an option for the treatment of Graves disease but is not an option for the acute treatment of thyroid storm.

👍 👎 Discuss (5) Improve

Next question

Thyroid storm ★

Thyroid storm is a rare but life-threatening complication of thyrotoxicosis. It is typically seen in patients with established thyrotoxicosis and is rarely seen as the presenting feature. Iatrogenic thyroxine excess does not usually result in thyroid storm.

Precipitating events:
- thyroid or non-thyroidal surgery
- trauma
- infection
- acute iodine load e.g. CT contrast media

Clinical features include:
- fever > 38.5ºC
- tachycardia
- confusion and agitation
- nausea and vomiting
- hypertension
- heart failure
- abnormal liver function test - jaundice may be seen clinically

Management:
- symptomatic treatment e.g. paracetamol
- treatment of underlying precipitating event
- beta-blockers: typically IV propranolol
- anti-thyroid drugs: e.g. methimazole or propylthiouracil
- Lugol's iodine
- dexamethasone - e.g. 4mg IV qds - blocks the conversion of T4 to T3

+ Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Media

THYROID
STORM

Thyroid storm

Osmosis - YouTube 👍 8 👎 1

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 2

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Score: **57%**

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A 34-year-old woman has been referred to your clinic with weight gain and irregular periods.

Observations: heart rate 95bpm, blood pressure 155/92 mmHg.

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
MCV	112 fl	(80-100)
Platelets	199 * 10^9/L	(150 - 400)
WBC	5.5 * 10^9/L	(4.0 - 11.0)
Bilirubin	17 µmol/L	(3 - 17)
ALP	138 u/L	(30 - 100)
ALT	55 u/L	(3 - 40)
9AM Cortisol	461 nmol/L	(140-690)
Midnight Cortisol	154 nmol/L	(140-690)
Cortisol following insulin stress test	645 nmol/L	(>170 rise compared to 9 AM cortisol)
Urine free cortisol	802 nmol/24h	(<120)
ACTH	13.3 pmol/L	(2.2-13.3)

MRI head: unremarkable intracranial appearances.

CT thorax abdomen pelvis: fatty changes within the liver with parenchymal heterogeneity, pelvic viscera unremarkable, clear lung fields.

What is the most likely diagnosis?

○ Cushing's disease ×

○ Cushing's syndrome ×

○ Exogenous steroid use ×

○ Hypothyroidism ×

○ Pseudo-Cushing's ×

Submit answer

Reference ranges ⌄

Score: **57%**

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ACTH	13.3 pmol/L	(2.2-13.3)

MRI head: unremarkable intracranial appearances.

CT thorax abdomen pelvis: fatty changes within the liver with parenchymal heterogeneity, pelvic viscera unremarkable, clear lung fields.

What is the most likely diagnosis?

Cushing's disease 17%

Cushing's syndrome 15%

Exogenous steroid use	13%
Hypothyroidism	7%
Pseudo-Cushing's	48%

The insulin tolerance test can be used to distinguish Cushing's syndrome from pseudo-Cushing's

Important for me Less important

In this case, we have a patient with cushingoid features - weight gain, hypertension, and irregular periods. Her blood tests show macrocytic anaemia and abnormal liver function tests which could be in keeping with alcohol excess. A CT scan showing fatty liver changes adds weight to this assumption. Imaging has not shown any obvious adenoma, ovarian cysts, or gland hypertrophy.

Pseudo-Cushing's is the correct answer. Pseudo-Cushing's is also known as 'physiological hypercortisolism'. This can be a result of pregnancy, polycystic ovarian syndrome, chronic alcohol abuse, depression, and severe physical stress (e.g. infection, surgery). In these cases, there is physiological over-activity of the hypothalamic-pituitary-adrenal axis in response to these states. It is difficult to differentiate it from Cushing's syndrome. Features of pseudo-Cushing's include; normal or mildly raised serum cortisol, maintained diurnal variation in serum cortisol, a raised urine free cortisol, and a rise in cortisol levels of >170nmol following an insulin stress test. Some Cushing's symptoms such as striae, easy bruising, and proximal myopathy are not usually seen in pseudo-Cushing's.

Cushing's disease is a particular type of Cushing's syndrome where a pituitary tumour causes high levels of cortisol due to increased ACTH secretion. This can be differentiated from adrenal Cushing's by checking ACTH levels (these would be high in Cushing's disease), finding a pituitary adenoma on MRI brain, CRH stimulation testing (in Cushing's disease you would see a high ACTH level), and inferior petrosal sinus sampling (high levels of ACTH in these samples compared to serum). In this case, Cushing's disease is not the correct answer as MRI scanning did not show a pituitary lesion, we would also expect to see a higher ACTH level.

Cushing's syndrome is a syndrome of cortisol excess. Unlike pseudo-Cushing's, this is not in response to a particular physiological state but is due to an abnormality in the hypothalamic-pituitary-adrenal axis. In Cushing's syndrome, you would expect to see high serum cortisol and high urine free cortisol, but generally to a higher degree than seen in this case. There would also be a loss of diurnal variation and the rise in cortisol in response to the insulin stress test would be blunted as the sustained hypercortisolism suppressed CRH secretion.

Exogenous steroid use is an important differential, good history taking is extremely important. In exogenous steroid use, we would expect to see low ACTH and low cortisol levels.

Hypothyroidism may present with the symptoms this patient has, but would not explain the abnormal urine free cortisol.

👍 | 👎 | 💬 Discuss (8) | Improve

Next question

Cushing's syndrome: investigations ★

This section will consider:
- the general lab findings consistent with Cushing's syndrome
- tests to confirm whether a patient indeed has Cushing's syndrome
- tests to find the underlying cause of Cushing's syndrome

It is useful to bear in mind the possible causes:
- iatrogenic: corticosteroid therapy
- ACTH-dependent causes
    - Cushing's disease (a pituitary adenoma → ACTH secretion)
    - ectopic ACTH secretion secondary to a malignancy
- ACTH-independent causes
    - adrenal adenoma

General findings consistent with Cushing's syndrome

A hypokalaemic metabolic alkalosis may be seen, along with impaired glucose tolerance.

Ectopic ACTH secretion (e.g. secondary to small cell lung cancer) is characteristically associated with very low potassium levels.

Tests to confirm Cushing's syndrome

The three most commonly used tests are:
- overnight (low-dose) dexamethasone suppression test
    - this is the most sensitive test and is now used first-line to test for Cushing's syndrome
    - patients with Cushing's syndrome do not have their morning cortisol spike suppressed
- 24 hr urinary free cortisol
    - two measurements are required
- bedtime salivary cortisol
    - two measurements are required

Localisation tests

The first-line localisation is 9am and midnight plasma ACTH (and cortisol) levels. If ACTH is suppressed then a non-ACTH dependent cause is likely such as an adrenal adenoma

High-dose dexamethasone suppression test

The high-dose dexamethasone suppression test may be used to localise the pathology resulting in Cushing's syndrome. This test may be interpreted as follows:

Cortisol	ACTH	Interpretation
Not suppressed	Suppressed	Cushing's syndrome due to other causes (e.g. adrenal adenomas)

Cortisol	ACTH	Interpretation
Suppressed	Suppressed	Cushing's disease (i.e. pituitary adenoma → ACTH secretion)
Not suppressed	Not suppressed	Ectopic ACTH syndrome

Other tests

CRH stimulation
- if pituitary source then cortisol rises
- if ectopic/adrenal then no change in cortisol

Petrosal sinus sampling of ACTH may be needed to differentiate between pituitary and ectopic ACTH secretion.

An insulin stress test is used to differentiate between true Cushing's and pseudo-Cushing's.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypothalamus
Anterior Pituitary
Adrenal Gland
Kidney
Dexamethasone 1mg
Cortisol
(Cushing's Syndrome)
ZERO TO FINALS

[Understanding The Dexamethasone Suppression Test](#)

Zero to Finals - YouTube 👍 31 👎 3

CUSHING SYNDROME

[Cushing syndrome](#)

Osmosis - YouTube 👍 7 👎 1

CUSHING'S SYNDROME

[Understanding Cushing's Syndrome](#)

Zero To Finals - YouTube 👍 1 👎 1

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A 35-year-old woman presents with weight gain for the last couple of years. She feels that most weight has been gained on her face and abdomen. On further questioning, her periods have been irregular for some time. She was diagnosed with type 2 diabetes mellitus last year. The patient drinks approximately 15 units of alcohol per day.

On examination, she has violaceous striae across the abdomen. Proximal muscle weakness is also evident when asking the patient to stand. Blood tests are taken:

Na^+	143 mmol/L	(135 - 145)
K^+	2.8 mmol/L	(3.5 - 5.0)

The team suspects that the patient's alcohol use may be the underlying cause of her presentation, rather than an endogenous cause, and want to differentiate the two.

What is the most appropriate investigation?

- ○ High-dose dexamethasone suppression test ×
- ○ Insulin stress test ×
- ○ Low-dose dexamethasone suppression test ×
- ○ Petrosal sinus sampling ×
- ○ Short synacthen test ×

Submit answer

Reference ranges

Score: **56.3%**

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K^+	2.8 mmol/L	(3.5 - 5.0)

The team suspects that the patient's alcohol use may be the underlying cause of her presentation, rather than an endogenous cause, and want to differentiate the two.

What is the most appropriate investigation?

High-dose dexamethasone suppression test	24%
🟩 Insulin stress test	41%
Low-dose dexamethasone suppression test	28%
Petrosal sinus sampling	2%
🟥 Short synacthen test	5%

The insulin tolerance test can be used to distinguish Cushing's syndrome from pseudo-Cushing's

Important for me Less important

This patient may have pseudo-Cushing's syndrome as a result of alcohol use, as opposed to true Cushing's syndrome. Patients with pseudo-Cushing's syndrome display clinical and biochemical features similar to those seen in Cushing's syndrome, such as the scenario here, but the presentation is caused by alcohol abuse, rather than an endogenous source or steroid use. Other causes of pseudo-Cushing's include severe depression, eating disorders, and chronic illness. Of the options listed above, the **insulin stress test** is used to differentiate between true Cushing's and pseudo-Cushing's.

The **high-dose dexamethasone suppression test** is used to differentiate pituitary from ectopic sources of Cushing's syndrome. It is not useful in pseudo-Cushing's.

The **low-dose dexamethasone suppression test** is used as a first-line test for patients with suspected Cushing's syndrome. It will not help to differentiate between Cushing's and pseudo-Cushing's.

Petrosal sinus sampling is performed for patients with confirmed ACTH-dependent Cushing's syndrome without an obvious pituitary lesion on MRI. Blood is sampled peripherally and from the inferior petrosal sinuses and the ratio of ACTH is measured. It is not useful to distinguish Cushing's from pseudo-Cushing's.

The **short synacthen test** is not relevant here. It is involved in the diagnosis of Addison's disease, rather than Cushing's syndrome.

Discuss (8) Improve

Next question

Cushing's syndrome: investigations

This section will consider:

- the general lab findings consistent with Cushing's syndrome
- tests to confirm whether a patient indeed has Cushing's syndrome
- tests to find the underlying cause of Cushing's syndrome

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+ Q 123

Next question

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Extended textbook

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Adrenal Gland
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ZERO TO FINALS

Understanding The Dexamethasone Suppression Test

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CUSHING SYNDROME

Cushing syndrome

Osmosis - YouTube 👍 7 👎 1

CUSHING'S SYNDROME

Understanding Cushing's Syndrome

Zero To Finals - YouTube 👍 1 👎 1

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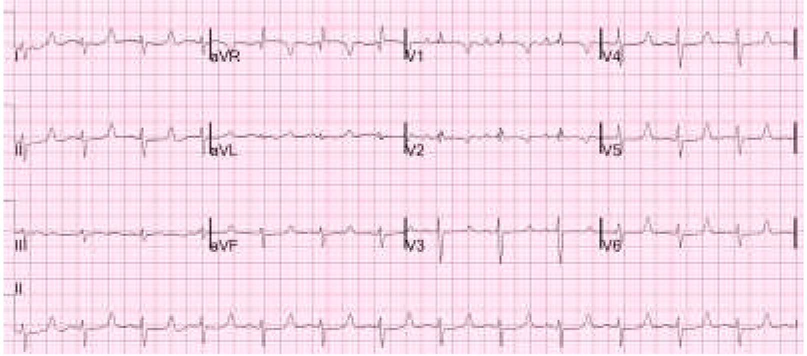
Back to top

A 40-year-old woman presents to the ambulatory clinic with a 4-day history of lethargy and weakness. There is also a history of intermittent cramps, particularly when trying to use her hands. She reports having had a recent gastrointestinal infection prior to the onset of her symptoms and has only just begun to eat and drink normally. Her past medical history consists of coeliac disease and vitiligo and her regular medications include folic acid and ferrous sulphate.

On examination, her observations are within normal limits. Her cardiorespiratory and abdominal examinations are unremarkable.

A set of routine blood tests are requested and an ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the most appropriate intervention to improve this patient's symptoms?

- ○ 0.9% sodium chloride with potassium supplementation
- ○ Calcium chloride
- ○ Hartmann's solution
- ○ Magnesium sulphate

○ Sodium bicarbonate ×

Submit answer

Reference ranges ⌄

Score: **55.7%**

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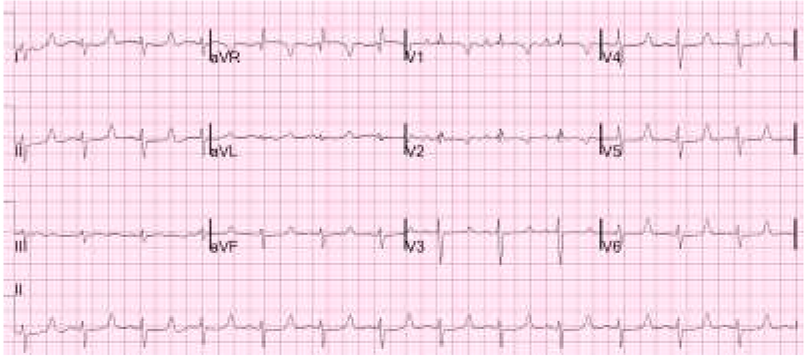


© Image used on license from Dr Smith, University of Minnesota

What is the most appropriate intervention to improve this patient's symptoms?

	0.9% sodium chloride with potassium supplementation	35%
🟩	Calcium chloride	37%
🟥	Hartmann's solution	2%
	Magnesium sulphate	23%

Sodium bicarbonate 2%

Calcium chloride is correct. This patient has a history of lethargy and muscle cramping along with an ECG that demonstrates a prolonged QT interval. A combination of these features is highly suspicious for hypocalcaemia. Given the past medical history of coeliac disease, it is quite possibly a malabsorptive cause of hypocalcaemia contributing to this patient's symptoms. In the context of significant hypocalcaemia, intravenous replacement should be given with either calcium chloride or calcium gluconate.

0.9% sodium chloride with potassium supplementation is incorrect. There are no features of hypokalaemia (e.g. U waves) on this patient's ECG.

Hartmann's solution is incorrect. There is nothing to suggest that this patient would benefit from intravenous fluid resuscitation.

Magnesium sulphate is incorrect. This is classically used in the management of Torsades de Pointes. However, it is also commonly given in the treatment of other arrhythmias (e.g. atrial fibrillation). Given that this patient has a prolonged QTc interval, and not yet an arrhythmia (such as Torsades de Pointes that is associated with QTc interval prolongation), the priority should be to reduce the QTc interval which is with the administration of intravenous calcium. Furthermore, calcium would also help to improve the patient's physical symptoms.

Sodium bicarbonate is incorrect. This is not indicated in the management of hypocalcaemia or QTc interval prolongation.

👍 👎 Discuss (10) Improve

Next question

Hypocalcaemia: features

As extracellular calcium concentrations are important for muscle and nerve function many of the features seen in hypocalcaemia seen a result of neuromuscular excitability

Features

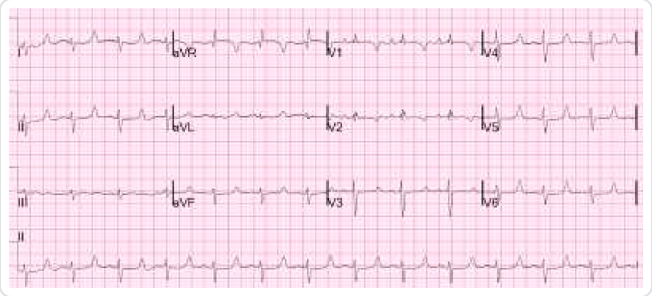
- tetany: muscle twitching, cramping and spasm
- perioral paraesthesia
- if chronic: depression, cataracts
- ECG: prolonged QT interval

Trousseau's sign

- carpal spasm if the brachial artery occluded by inflating the blood pressure cuff and maintaining pressure above systolic
- wrist flexion and fingers are drawn together
- seen in around 95% of patients with hypocalcaemia and around 1% of normocalcaemic people

Chvostek's sign

- tapping over parotid causes facial muscles to twitch
- seen in around 70% of patients with hypocalcaemia and around 10% of normocalcaemic people



+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

HYPOCALCEMIA

Hypocalcemia

Osmosis - YouTube 👍 2 👎 0

Report broken media

Score: **55.1%**

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A 19-year-old girl was seen in clinic with lethargy, weakness worsening over the past 4 weeks. She also complains of recurrent muscle cramps in her legs, causing her to have trouble sleeping. On further questioning she admits to urinary frequency, passing urine up to ten times a day, and feels dehydrated all the time. She also mentions that her periods which were usually irregular, have stopped 4 months ago.

On examination, she is thin, with a body mass index of 17kg/m². Her heart rate is 88 bpm and blood pressure is 108/86 mmHg.

C Reactive protein	2mg/l
Haemoglobin	158 g/l
White cell count	7.6 x 10^9/L
Na+	136 mmol/l
K+	2.9 mmol/l
Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.42 mmol/l

Venous blood gas result

pH	7.532
Bicarbonate	37mmol/l

What would be the next most useful investigation?

○ Transvaginal ultrasound (TVUS) of the ovaries ×

○ Urine diuretic assay ×

○ Early morning cortisol ×

○ Serum renin and aldosterone levels ×

○ Fasting blood glucose levels ×

Submit answer

Reference ranges

Score: **55.1%**

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Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.42 mmol/l

Venous blood gas result

pH	7.532
Bicarbonate	37mmol/l

What would be the next most useful investigation?

Transvaginal ultrasound (TVUS) of the ovaries	4%
[✓ Urine diuretic assay]	36%
Early morning cortisol	20%

Serum renin and aldosterone levels	34%
Fasting blood glucose levels	6%

Patients with hypokalaemia, metabolic alkalosis and a normal - low blood pressure the following differentials should be considered - diuretic abuse, Bartter's syndrome, Gitelman's syndrome. Of the three, the most common cause is diuretic abuse, especially in young women, and can be ruled out with a urine diuretic assay.

Bartter's syndrome presents early in life, with classical features of triangular facies, polyuria, polydipsia and renal failure. Serum renin and aldosterone levels are high despite a low or normal blood pressure. Urine calcium may be raised, and renal stones are a common feature. In Gitelman's syndrome patients may present later on in adulthood, but have a milder disease course or may be asymptomatic compared to patients with Bartter's syndrome. Hypomagnesaemia and hypocalciuria differentiates Gitelman's syndrome from Bartter's syndrome.

This patient may need other further investigations such as a TVUS, early morning cortisol and fasting blood glucose tests to rule out other conditions, but in view of her biochemistry profile, a urine diuretic assay would be the most useful next investigation to perform.

👍 👎 Discuss (9) Improve

Next question

Hypokalaemia

Potassium and hydrogen can be thought of as competitors. Hyperkalaemia tends to be associated with acidosis because as potassium levels rise fewer hydrogen ions can enter the cells

Hypokalaemia with alkalosis
- vomiting
- thiazide and loop diuretics

- Cushing's syndrome
- Conn's syndrome (primary hyperaldosteronism)

Hypokalaemia with acidosis
- diarrhoea
- renal tubular acidosis
- acetazolamide
- partially treated diabetic ketoacidosis

Magnesium deficiency may also cause hypokalaemia. In such cases, normalizing the potassium level may be difficult until the magnesium deficiency has been corrected

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Hypokalemia - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube 👍 4 👎 1

Report broken media

Score: **54.4%**

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A 53 year old gentleman attends his General Practitioner for a 'Well Man Check'. He has a past medical history of hypertension which has been treated with ramipril for 4 years. As part of the screening the GP notes that the patient has been suffering from low back pain for the last couple of months. He has been taking paracetamol and ibuprofen that he has bought over the counter and this has eased his pain. His blood pressure today is 134/76 mmHg. His GP takes blood tests as part of the check and the results are shown below.

Hb	13.2 g/dl
Platelets	312 * 10^9/l
WBC	8.2 * 10^9/l

Na^+	138 mmol/l
K^+	6.6 mmol/l
Urea	6.2 mmol/l
Creatinine	114 µmol/l

His GP notifies the patient immediately on seeing these results and refers him to the local Medical Assessment Unit. Where more tests are carried out.

Arterial Blood Gases:

pH 7.34
PaCO2 5.1kPa
PaO2 12kPa
HCO3- 20 mmol/l

Serum Chloride 120mmol/l

Urinalysis:

pH 4.8
Protein negative
Blood negative
Leukocytes negative
Glucose negative

What is the most likely diagnosis?

Renal Tubular Acidosis type 1	6%
Renal Tubular Acidosis type 2	5%
Renal Tubular Acidosis type 3	2%
[✓ Renal Tubular Acidosis type 4]	78%
NSAID induced nephropathy	9%

This patient has RTA type 4. This often occurs asymptomatically as in this case. In this case it's aetiology is likely secondary to ibuprofen use which has caused an aldesterone resistance in the proximal tubule of the nephron. It is associated with a mild metabolic acidosis and classically presents in patients with a high serum potassium. Urinary pH is commonly normal, but given that the nephron has not lost its hydrogen ion buffering capacity there is scope for the urinary pH to become more acidic to buffer the metabolic acidosis. The serum chloride level is high in this patient, with hyperchloraemia being a cardinal feature of all sub-types of RTA. When calculated, the anion gap in RTA type 4 is normal.

In NSAID induced nephropathy the urinalysis would show white cells (leukocytes) and may even show proteinuria.

👍 👎 Discuss (6) Improve

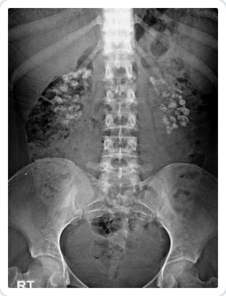
Next question

Renal tubular acidosis ★

All three types of renal tubular acidosis (RTA) are associated with hyperchloraemic metabolic acidosis (normal anion gap).

Type 1 RTA (distal)

- inability to generate acid urine (secrete H+) in distal tubule
- causes hypokalaemia
- complications include nephrocalcinosis and renal stones
- causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, analgesic nephropathy



Type 2 RTA (proximal)

- decreased HCO3- reabsorption in proximal tubule
- causes hypokalaemia
- complications include osteomalacia
- causes include idiopathic, as part of Fanconi syndrome, Wilson's disease, cystinosis, outdated tetracyclines, carbonic anhydrase inhibitors (acetazolamide, topiramate)

Type 3 RTA (mixed)

- extremely rare
- caused by carbonic anhydrase II deficiency
- results in hypokalaemia

Type 4 RTA (hyperkalaemic)

- reduction in aldosterone leads in turn to a reduction in proximal tubular ammonium excretion
- causes hyperkalaemia

- causes include hypoaldosteronism, diabetes

＋ Q 123

Next question

Textbooks

High-yield textbook

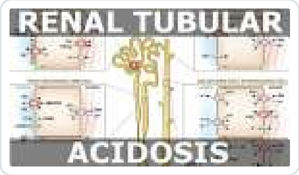
Extended textbook

Media

RENAL TUBULAR ACIDOSIS

Renal tubular acidosis

Osmosis - YouTube 👍 8 👎 0


RENAL TUBULAR
ACIDOSIS

Renal tubular acidosis

DirtyUSMLE - YouTube 👍 41 👎 4

Report broken media

Score: **54.9%**

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Question 92 of 101

An 18-year-old male with no prior medical history is admitted to the resuscitation room of Emergency Department where you are asked to assess him. He is noted to be hypotensive, tachycardic and febrile. Further assessment highlights a widespread, non-blanching, purple rash. He is diagnosed with meningococcal septicaemia and is treated appropriately. Nevertheless, his condition deteriorates further when he also develops Waterhouse-Friderichsen syndrome. Which of the following sets of blood results would be most consistent with his condition at this point?

○ Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 7.5mmol/L ✕

○ Na+ 147mmol/L, K+ 3.0mmol/L, Glucose 2.0mmol/L ✕

○ Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L ✕

○ Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L ✕

○ Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L ✕

Submit answer

Reference ranges

Score: **54.9%**

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Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 7.5mmol/L	1%
Na+ 147mmol/L, K+ 3.0mmol/L, Glucose 2.0mmol/L	8%
Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L	83%
Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L	2%
Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L	6%

Waterhouse-Friderichsen syndrome is defined as adrenal failure due to bleeding into the adrenal glands (otherwise referred to as haemorrhagic adrenalitis) and is most commonly caused by meningococcal septicaemia.

The biochemical pattern which results is analogous to that seen in an Addisonian crisis, and so you would expect to see hyponatraemia, hyperkalaemia and hypoglycaemia

Discuss (3) | Improve

Next question

Waterhouse-Friderichsen syndrome ★

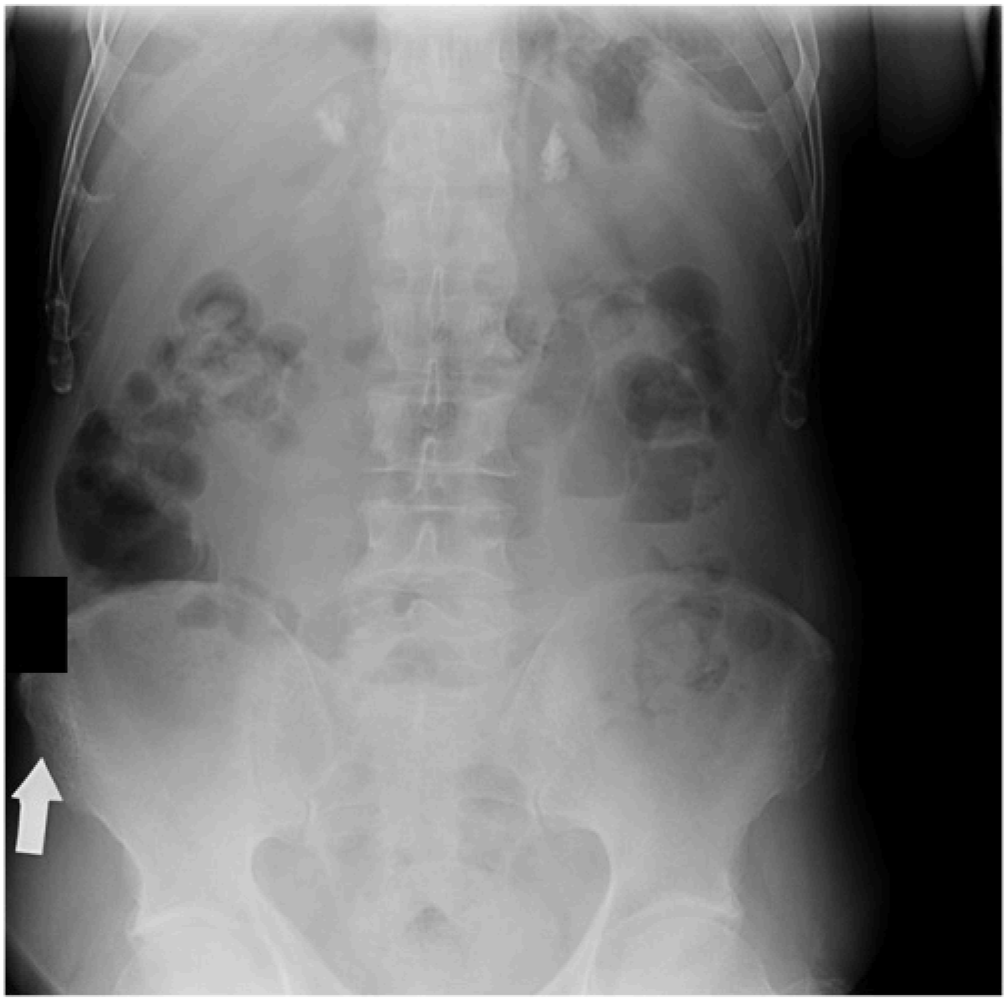
Waterhouse-Friderichsen syndrome describes adrenal gland failure secondary to a previous adrenal haemorrhage that was caused by severe bacterial infection.

Causes

- *Neisseria meningitidis*: most common cause
- *Haemophilus influenzae*
- *Pseudomonas aeruginosa*
- *Escherichia coli*
- *Streptococcus pneumoniae*

The features are those of hypoadrenalism, i.e.:

- lethargy, weakness, anorexia, nausea & vomiting, weight loss
- hyperpigmentation (especially palmar creases), vitiligo, loss of pubic hair in women
- crisis: collapse, shock, pyrexia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Calcification of the adrenal glands secondary to previous adrenal haemorrhage

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **55.4%**

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Question 93 of 101

You are asked to review a patient with hyperglycaemia on the ward. The patient is a 58-year-old female with type 1 diabetes mellitus. The nurse states that her capillary blood glucose is 12.8 mmol/l. Her drug history includes Humulin M3 (28 units before breakfast, and 38 units before evening meal). She had her normal insulin with breakfast at 07:30.

You review her at 11:30. The patient denies polyuria or polydipsia. She is clinically euvolaemic. She is medically stable and awaiting discharge from physiotherapy. You ask the nurse to check plasma ketones which return at 0.4mmol/l. On review of her insulin chart, you note that her blood glucose levels are usually well controlled.

How will you manage this patient?

- ○ 6 units of actrapid
- ○ Reassurance to the patient and nurse and continue monitoring
- ○ Intravenous insulin sliding scale
- ○ Diabetic ketoacidosis protocol
- ○ Increase Humulin M3 to 32 units before breakfast starting the following day

Submit answer

Reference ranges

Score: **55.4%**

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You are asked to review a patient with hyperglycaemia on the ward. The patient is a 58-year-old female with type 1 diabetes mellitus. The nurse states that her capillary blood glucose is 12.8 mmol/l. Her drug history includes Humulin M3 (28 units before breakfast, and 38 units before evening meal). She had her normal insulin with breakfast at 07:30.

You review her at 11:30. The patient denies polyuria or polydipsia. She is clinically euvolaemic. She is medically stable and awaiting discharge from physiotherapy. You ask the nurse to check plasma ketones which return at 0.4mmol/l. On review of her insulin chart, you note that her blood glucose levels are usually well controlled.

How will you manage this patient?

	6 units of actrapid	8%
🟩	Reassurance to the patient and nurse and continue monitoring	64%
	Intravenous insulin sliding scale	5%
	Diabetic ketoacidosis protocol	1%
🟥	Increase Humulin M3 to 32 units before breakfast starting the following day	22%

When managing hyperglycaemia in an insulin dependent diabetic, avoid the use of stat insulin, unless the patient is symptomatic or has high ketones (>0.6 mmol/l). It is safer to increase the 'usual' insulin if persistently hyperglycaemic

Important for me Less important

The patient has had a once off 'blip' in blood glucose control. When managing hyperglycaemia in an insulin dependent diabetic, you should avoid the use of stat insulin, unless the patient is symptomatic or has high ketones (>0.6 mmol/l). It is far safer to increase the 'usual' insulin if persistently hyperglycaemic.

The patient is medically stable, asymptomatic, and the ketones are at a satisfactory level. Therefore the patient and nurse should be reassured, and the blood glucose level monitored.

If the patient had frequent high blood sugars then it would be appropriate to titrate the normal insulin up.

An intravenous insulin sliding scale is indicated when the patient is undergoing fasting and will be missing more than one meal.

The diabetic ketoacidosis (DKA) protocol should be implemented in patients with DKA which is diagnosed when ketones are significantly elevated (> 3 mmol/l) and the patient is acidotic (HCO3 < 15).

👍 👎 Discuss (5) Improve

Next question

Insulin ★

Insulin is a peptide hormone, produced by beta cells of the pancreas, and is central to regulating carbohydrate and fat metabolism in the body. Insulin causes cells in the liver, skeletal muscles, and fat tissue to absorb glucose from the blood. In the liver and skeletal muscles, glucose is stored as glycogen, and in fat cells (adipocytes) it is stored as triglycerides.

Structure

The human insulin protein is composed of 51 amino acids, and has a molecular weight of 5808 Da. It is a dimer of an A-chain and a B-chain, which are linked together by disulfide bonds.

Synthesis

Pro-insulin is formed by the rough endoplasmic reticulum in pancreatic beta cells. Then pro-insulin is cleaved to form insulin and C-peptide. Insulin is stored in secretory granules and released in response to Ca^{2+}.

Function

Basics
- Secreted in response to hyperglycaemia
- Glucose utilisation and glycogen synthesis
- Inhibits lipolysis
- Reduces muscle protein loss
- Increases cellular uptake of potassium (via stimulation of Na+/K+ ATPase pump)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **54.8%**

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Question 94 of 101

A 29-year-old woman comes to the clinic with feelings of anxiety, palpitations and a resting tremor present over the past 2 weeks. She also has symptoms of a flu-like illness and pain over her anterior neck. On examination,you reveal tenderness over the thyroid. Her blood pressure is 115/88 mmHg, she has a fine tremor at rest, sweaty palms and a tachycardia of 88 beats per minute. TSH is <0.05 U/ml.

Which of the following is the most appropriate intervention?

- ○ Thyroxine ×
- ○ Carbimazole ×
- ○ Propylthiouracil ×
- ○ Carbimazole and thyroxine ×
- ○ Propranolol ×

Submit answer

Reference ranges

Score: **54.8%**

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A 29-year-old woman comes to the clinic with feelings of anxiety, palpitations and a resting tremor present over the past 2 weeks. She also has symptoms of a flu-like illness and pain over her anterior neck. On examination,you reveal tenderness over the thyroid. Her blood pressure is 115/88 mmHg, she has a fine tremor at rest, sweaty palms and a tachycardia of 88 beats per minute. TSH is <0.05 U/ml.

Which of the following is the most appropriate intervention?

	Thyroxine	1%
🟥	Carbimazole	13%
	Propylthiouracil	6%
	Carbimazole and thyroxine	5%
🟩	Propranolol	75%

The short history of flu-like symptoms, coupled with pain over the thyroid gland is consistent with a diagnosis of subacute thyroiditis. The presentation with thyrotoxicosis is caused by a transient increase in thyroid hormone release, rather than over production, hence anti-thyroid drugs are not indicated. Symptomatic relief with propranolol is indicated, as are non-steroidal anti-inflammatory agents for management of pain and inflammation. A period of hypothyroidism may follow, with later recovery to normal thyroid function.

Anti-thyroid drugs are not needed, carbimazole and propylthiouracil are therefore incorrect, as is the block-replace regimen option. Although a transient period of hypothyroidism may be seen after the thyrotoxicosis subsides, thyroxine replacement is usually not needed.

👍 👎 Discuss (3) Improve

Next question

Subacute (De Quervain's) thyroiditis

Subacute thyroiditis (also known as De Quervain's thyroiditis and subacute granulomatous thyroiditis) is thought to occur following viral infection and typically presents with hyperthyroidism.

There are typically 4 phases;

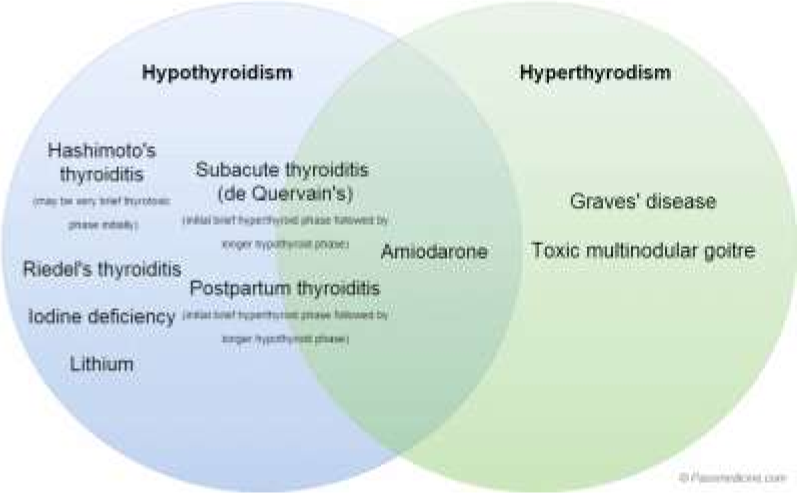
- phase 1 (lasts 3-6 weeks): hyperthyroidism, painful goitre, raised ESR
- phase 2 (1-3 weeks): euthyroid
- phase 3 (weeks - months): hypothyroidism
- phase 4: thyroid structure and function goes back to normal

Investigations

- thyroid scintigraphy: globally reduced uptake of iodine-131

Management

- usually self-limiting - most patients do not require treatment
- thyroid pain may respond to aspirin or other NSAIDs
- in more severe cases steroids are used, particularly if hypothyroidism develops


Hypothyroidism
Hyperthyrodism
Hashimoto's thyroiditis
(may be very brief thyrotoxic phase initially)
Subacute thyroiditis (de Quervain's)
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Amiodarone
Riedel's thyroiditis
Postpartum thyroiditis
Iodine deficiency
(initial brief hyperthyroid phase followed by longer hypothyroid phase)
Lithium
Graves' disease
Toxic multinodular goitre
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Venn diagram showing how different causes of thyroid dysfunction may manifest. Note how many causes of hypothyroidism may have an initial thyrotoxic phase.

Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **54.3%**

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Back to top

Question 95 of 101

A 53-year-old woman is referred to an endocrinology clinic. She has a history of Grave's disease which had previously been controlled on carbimazole however recently presented to her general practitioner due to a two-month history of diarrhoea, palpitations, and heat intolerance. She is post-menopausal and has not had any recent illness or relevant past medical history. The general practitioner performed some thyroid function tests, results of which are as follows:

TSH	0.2	(0.5-5.5 mu/L)
Free T4	25.2	(9-18 pmol/L)

On examination in the clinic, she is warm, tachycardic but otherwise well. A smooth, non-tender goitre is noted however there are no eye signs.

What is the most appropriate treatment?

- ○ Advice on medication compliance ✕
- ○ Prednisolone ✕
- ○ Propylthiouracil ✕
- ○ Radioiodine therapy ✕
- ○ Thyroidectomy ✕

Submit answer

Reference ranges

Score: **54.3%**

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TSH	0.2	(0.5-5.5 mu/L)
Free T4	25.2	(9-18 pmol/L)

On examination in the clinic, she is warm, tachycardic but otherwise well. A smooth, non-tender goitre is noted however there are no eye signs.

What is the most appropriate treatment?

🟥	Advice on medication compliance	17%
	Prednisolone	4%
	Propylthiouracil	14%
🟩	Radioiodine therapy	60%
	Thyroidectomy	4%

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Important for me Less important

Given this woman's medical history, findings of a smooth non-tender goitre, and laboratory results, the most likely diagnosis here is a relapse of her Grave's disease. In these cases and the absence of any contraindications, radioiodine therapy is the preferred treatment.

Prednisolone can be used in subacute thyroiditis however this is not the diagnosis here as evidenced by the lack of recent illness and non-tender goitre.

Propylthiouracil is an alternative antithyroid drug to carbimazole and is preferred in early pregnancy. As this woman is presenting with a relapse of Grave's disease, radioiodine therapy is preferred. Also, note she is post-menopausal, and therefore pregnancy is not a contraindication here to either radioiodine therapy or carbimazole.

Thyroidectomy is not the first-line treatment in a relapse of previously well-controlled Grave's disease.

👍 👎 Discuss (6) Improve

Next question

Graves' disease: management

Despite many trials, there is no clear guidance on the optimal management of Graves' disease. Treatment options include anti-thyroid drugs (ATDs, for example carbimazole), radioiodine treatment and surgery.

Anti-thyroid drugs have emerged as the most popular first-line therapy for Graves' disease in recent years. Factors that particularly support their use include anti-thyroid drugs significant symptoms of thyrotoxicosis, or patients with a significant risk of hyperthyroid complications (e.g. elderly patients, cardiovascular disease).

Initial treatment to control symptoms
- propranolol is used to help block the adrenergic effects

NICE recommends that patients with Graves' disease are referred to secondary care for ongoing treatment.

- NICE suggest carbimazole should be considered in primary care if patients symptoms are not controlled with propanolol

ATD therapy

- carbimazole is started at 40mg and reduced gradually to maintain euthyroidism
- typically continued for 12-18 months
- the major complication of carbimazole therapy is agranulocytosis
- an alternative regime is termed 'block-and-replace'
  - carbimazole is started at 40mg
  - thyroxine is added when the patient is euthyroid
  - treatment typically lasts for 6-9 months
  - patients following an ATD titration regime have been shown to suffer fewer side-effects than those on a block-and-replace regime

Radioiodine treatment

- often used in patients who relapse following ATD therapy or are resistant to primary ATD treatment
- contraindications include pregnancy (should be avoided for 4-6 months following treatment) and age < 16 years. Thyroid eye disease is a relative contraindication, as it may worsen the condition
- the proportion of patients who become hypothyroid depends on the dose given, but as a rule the majority of patient will require thyroxine supplementation after 5 years

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 11 👎 4

Graves' disease: management

Suggest link Report broken link

Media

HYPERTHYROIDISM
TREATMENT MEDICATIONS
Osmosis.org

Hyperthyroidism treatment medications

Osmosis - YouTube 👍 5 👎 0

ENDOCRINOLOGY
GRAVES DISEASE

Graves' disease

Armando Hasudungan - YouTube 👍 2 👎 2

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A surgical Foundation Year 1 doctor (FY1) asks you to review a preoperative ECG for a 19-year-old patient who has been admitted under their team with suspected appendicitis. The only abnormality is a prolonged QT and you note the adjusted calcium to be 2.02 mmol/l.

The FY1 tells you that when they looked at the patients closed fists the outer two knuckles looked like dimples. She also tells you that the patient's body mass index is 29 kg/m².

You ask her to order some blood tests which come back as follows:

Adjusted calcium	2.02 mmol/l
PTH	69 pmol/L (normal range = 0.8 - 8.5)
Phosphate	2.0 mmol/l
ALP	130 u/l

What is the most likely underlying cause for this patient's hypocalcaemia?

- ○ Hypoparathyroidism ✕
- ○ Pseudohypoparathyroidism type 1a ✕
- ○ Pseudohypoparathyroidism type 1b ✕
- ○ Pseudopseudohypoparathyroidism ✕
- ○ Secondary hyperparathyroidism ✕

Submit answer

Reference ranges

Score: **53.7%**

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You ask her to order some blood tests which come back as follows:

Adjusted calcium	2.02 mmol/l
PTH	69 pmol/L (normal range = 0.8 - 8.5)
Phosphate	2.0 mmol/l
ALP	130 u/l

What is the most likely underlying cause for this patient's hypocalcaemia?

Hypoparathyroidism	2%
Pseudohypoparathyroidism type 1a	52%
Pseudohypoparathyroidism type 1b	12%
Pseudopseudohypoparathyroidism	16%
Secondary hyperparathyroidism	18%

Short fifth metacarpals, short stature, learning difficulties + ↑ PTH, ↓ calcium, ↑ phosphate → pseudohypoparathyroidism

Important for me Less important

This patient has a high PTH, a low calcium, a high phosphate and a normal ALP. The patient is also obese and the dimples on the outer two knuckles are likely to represent shortening of the 4th and 5th metacarpals. This biochemistry in combination of these clinical features is characteristic of pseudohypoparathyroidism Type 1a (Albright's Hereditary Osteodystrophy).

Pseudopseudohypoparathyroidism would have the same clinical features but would have normal biochemistry.
Pseudohypoparathyroidism Type 1b would have the same biochemistry but lack the clinical features.

This patient has a high PTH, therefore this immediately excludes hypoparathyroidism. In secondary hyperparathyroidism the ALP would be elevated therefore this is incorrect.

👍 👎 Discuss (13) Improve

Next question

Pseudohypoparathyroidism ★

Pseudohypoparathyroidism is caused by target cell insensitivity to parathyroid hormone (PTH) due to a mutation in a G-protein. In type I pseudohypoparathyroidism there is a complete receptor defect whereas in type II the cell receptor is intact. Pseudohypoparathyroidism is typically inherited in an autosomal dominant fashion.

Bloods

- ↑ PTH
- ↓ calcium
- ↑ phosphate

Features

- short fourth and fifth metacarpals
- short stature
- learning difficulties

- obesity
- round face

Investigation

- an infusion of PTH followed by measurement of urinary phosphate and cAMP measurement - this can help differentiate between type I (neither phosphate or cAMP levels rise) and II (cAMP rises but phosphate levels do not change)

+ Q 123

Next question

B *I* **A** T↕

Textbooks

High-yield textbook

Extended textbook

Score: **54.2%**

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Back to top

A 28-year-old pregnant woman at eight weeks gestation with a history of type 1 diabetes mellitus attends a routine check-up. She takes a basal/bolus insulin regimen and reports no symptoms of hypoglycaemia. On examination, her blood pressure is 110/80 mmHg, pulse rate is 78 beats per minute, and body mass index (BMI) is 25 kg/m². Her latest glycated haemoglobin (HbA1c) level is 45 mmol/mol. The results of her investigations are as follows:

Hb	120 g/L	Female: (115 - 160)
Platelets	290 * 10^9/L	(150 - 400)
WBC	8.5 * 10^9/L	(4.0 - 11.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
ALT	38 u/L	(3 - 40)
Albumin	41 g/L	(35 - 50)

What is the most appropriate advice for this patient?

- ○ Add vildagliptin ×
- ○ Folic acid supplementation 0.5 mg/day ×
- ○ Increased frequency of retinal screening ×
- ○ Refer for detailed anomaly scan ×
- ○ Weight loss ×

Submit answer

Reference ranges ⌵

Score: **54.2%**

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Creatinine	98 µmol/L	(55 - 120)
ALT	38 u/L	(3 - 40)
Albumin	41 g/L	(35 - 50)

What is the most appropriate advice for this patient?

	Add vildagliptin	1%
🟥	Folic acid supplementation 0.5 mg/day	34%
🟩	Increased frequency of retinal screening	45%
	Refer for detailed anomaly scan	17%
	Weight loss	4%

Patients with diabetes should have increased frequency of retinal screening during pregnancy due to increased risk of retinopathy

Important for me Less important

Increased frequency of retinal screening is the correct answer. Women who are pregnant with pre-existing diabetes are at an elevated risk of worsening retinopathy; therefore, they require more frequent retinal screenings. These screenings must be conducted alongside meticulous glycaemic control to mitigate the risk of complications.

Add vildagliptin is incorrect. The patient's HbA1C level is below 48 mmol/mol, which falls within the target range during pregnancy, thus negating the necessity for additional pharmacotherapy. Furthermore, metformin is the only oral anti-diabetic medication recommended during pregnancy alongside insulin; other oral hypoglycaemic agents should be discontinued.

Folic acid supplementation 0.5 mg/day is not advised as per current guidelines; a higher dosage of 5mg/day until the 12th week of gestation is typically prescribed to ensure adequate folate levels. This higher dose is particularly important in pregnant women with pre-existing diabetes to reduce the risk of neural tube defects.

Refer for detailed anomaly scan is customarily performed at around 20 weeks of gestation. At eight weeks of gestation, conducting this type of scan would be premature.

Weight loss advice is generally reserved for women with a BMI exceeding 27 kg/m². Given that this patient's BMI is 25 kg/m², recommending weight loss would be inappropriate.

👍 👎 Discuss (2) Improve

Next question

Gestational diabetes ★

Diabetes mellitus may be a pre-existing problem or develop during pregnancy, gestational diabetes. It complicates up to 1 in 20 pregnancies. NICE estimates the following breakdown:

- 87.5% have gestational diabetes
- 7.5% have type 1 diabetes

- 5% have type 2 diabetes

Gestational diabetes

Gestational diabetes is the second most common medical disorder complicating pregnancy (after hypertension), affecting around 4% of pregnancies.

Risk factors for gestational diabetes

- BMI of > 30 kg/m²
- previous macrosomic baby weighing 4.5 kg or above
- previous gestational diabetes
- first-degree relative with diabetes
- family origin with a high prevalence of diabetes (South Asian, black Caribbean and Middle Eastern)
- unexplained stillbirth in a previous pregnancy

Screening for gestational diabetes

- the oral glucose tolerance test (OGTT) is the test of choice
- women who've previously had gestational diabetes: OGTT should be performed as soon as possible after booking and at 24-28 weeks if the first test is normal. NICE also recommend that early self-monitoring of blood glucose is an alternative to the OGTTs
- women with any of the other risk factors should be offered an OGTT at 24-28 weeks

Diagnostic thresholds for gestational diabetes

- these have recently been updated by NICE, gestational diabetes is diagnosed if either:
- fasting glucose is >= 5.6 mmol/L
- 2-hour glucose is >= 7.8 mmol/L

Management of gestational diabetes

- newly diagnosed women should be seen in a joint diabetes and antenatal clinic within a week
- women should be taught about self-monitoring of blood glucose

- advice about diet (including eating foods with a low glycaemic index) and exercise should be given
- if the fasting plasma glucose level is < 7 mmol/l a trial of diet and exercise should be offered
  - if glucose targets are not met within 1-2 weeks of altering diet/exercise metformin should be started
  - if glucose targets are still not met insulin should be added to diet/exercise/metformin
  - gestational diabetes is treated with short-acting, not long-acting, insulin
- if at the time of diagnosis the fasting glucose level is >= 7 mmol/l insulin should be started
- if the plasma glucose level is between 6-6.9 mmol/l, and there is evidence of complications such as macrosomia or hydramnios, insulin should be offered
- glibenclamide should only be offered for women who cannot tolerate metformin or those who fail to meet the glucose targets with metformin but decline insulin treatment

Management of pre-existing diabetes

- weight loss for women with BMI of > 27 kg/m^2
- stop oral hypoglycaemic agents, apart from metformin, and commence insulin
- folic acid 5 mg/day from pre-conception to 12 weeks gestation
- detailed anomaly scan at 20 weeks including four-chamber view of the heart and outflow tracts
- tight glycaemic control reduces complication rates
- treat retinopathy as can worsen during pregnancy

Targets for self monitoring of pregnant women (pre-existing and gestational diabetes)

Time	**Target**
Fasting	5.3 mmol/l
1 hour after meals	7.8 mmol/l, or:
2 hour after meals	6.4 mmol/l

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 12 👎 16

DKA in pregnancy

NICE 👍 12 👎 13

2015 Management of diabetes in pregnancy

Suggest link

Report broken link

Media

Gestational Diabetes

Armando Hasudungan - YouTube 👍 3 👎 2

Report broken media

Score: **53.6%**

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97 ✗

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← Question 98 of 101 ✗ ⚐ →

A 42-year-old man presents to endocrinology clinic. He has been seeing his GP due to developing gynaecomastia. At first, this was thought to be due to alcohol excess and possible liver involvement, but after stopping alcohol completely and normal liver function tests this diagnosis was excluded. He has no previous medical history apart from having had a tibial fracture a year ago, and having been diagnosed with migraines two months ago. He takes paracetamol for the migraines but finds this to be not very helpful, especially with the headaches at night. On more direct questioning he has also developed problems maintaining an erection. On examination he does have gynaecomastia, but apart from this he appears normal. On repeating blood tests in the clinic his liver function tests are normal, but morning serum testosterone is low, with normal FSH and LH. What further investigation is likely to be diagnostic?

	Liver biopsy	1%
	Transferrin saturations	8%
🟩	Serum prolactin	71%
🟥	CT brain	13%
	Thyroid function tests	7%

Hypogonadism secondary to prolactinaemia would be associated with low testosterone and low or normal FSH and LH

Important for me Less important

This patient has symptoms of hypogonadism with erectile dysfunction and gynaecomastia with a suggested evidence of a headache suspicious for an intracranial lesion, especially with a new diagnosis of headaches at night. The most likely explanation is a prolactinoma, which can be detected by very high prolactin levels. After this, an MRI of the pituitary would be appropriate. A CT brain is unlikely to be able to demonstrate any lesions. A liver biopsy could be considered if there was liver failure and it was unexplained. Transferrin saturation could be considered if haemochromatosis was considered.

Discuss (10) Improve

Next question

Hypogonadism in men

Causes of hypogonadism in men
- primary (disease of the testes)
  - childhood mumps
- secondary (disease of the hypothalamus or pituitary)
  - Klinefelter syndrome
  - Kallman syndrome

Testosterone levels in men also tend to decline with age.

Testosterone deficiency causes a number of changes in men:
- loss of libido
- erectile dysfunction
- lethargy
- decreased muscle mass and strength
- reduced facial hair growth
- impaired glucose tolerance

+ Q 123

Next question

B I A

Dr. Jamil Alrajhi

Textbooks

High-yield textbook

Extended textbook

Media

KLINEFELTER SYNDROME

Klinefelter syndrome

Osmosis - YouTube 👍 6 👎 1

KALLMANN SYNDROME

Kallman's syndrome

Osmosis - YouTube 👍 5 👎 1

Report broken media

Score: **53.1%**

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A 35-year-old woman is reviewed in endocrinology outpatient clinic. She reports a three-month history of headaches, palpitations, mild constipation, and urinary frequency. She is persistently hypertensive despite treatment with the maximum dose of lisinopril, amlodipine, and indapamide.

Her observations are:

- Respiratory rate: 18/min
- Oxygen saturation: 99% on room air
- Heart rate: 115bpm
- Blood pressure: 187/98mmHg
- Temperature: 36.2 ^{o}C
- GCS: 15/15

An ECG is performed which shows sinus tachycardia.

A set of blood tests has been performed. The results are as follows:

Hb	140 g/L	Female: (115 - 160)
Platelets	387 * 10^9/L	(150 - 400)
WBC	9.0 * 10^9/L	(4.0 - 11.0)

Calcium	2.80 mmol/L	(2.1-2.6)
Phosphate	1.2 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)
Thyroid-stimulating hormone (TSH)	5.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	12 pmol/L	(9.0 - 18)
Parathyroid hormone	52 pg/mL	(10 - 55)

Given the likely diagnosis, what is the most appropriate blood pressure treatment?

	Atenolol	2%
	Bisoprolol	5%
	Nicorandil	2%
🟩	Phenoxybenzamine	78%
🟥	Spironolactone	12%

PHaeochromocytoma - give **PH**enoxybenzamine before beta-blockers

Important for me Less important

This patient has symptoms of hypercalcaemia (constipation, urinary frequency), coupled with a mildly raised serum calcium level. The serum parathyroid hormone (PTH) level is at the upper end of normal - this is suggestive of primary hyperparathyroidism. This patient has several cardinal symptoms of a phaeochromocytoma (headache, palpitations, hypertension). This patient is also likely to have a phaeochromocytoma, as the combination of primary hyperparathyroidism (secondary to a parathyroid adenoma or parathyroid hyperplasia) and a phaeochromocytoma is in keeping with multiple endocrine neoplasia type 2 (MEN-2A). The patient will require cross-sectional imaging to localise the phaeochromocytoma. Treatment should be initiated in the interim to control symptoms.

Phenoxybenzamine is the correct answer. Alpha-blockers should be initiated first-line. Initiation of beta-blockers before alpha-blockade will cause unopposed stimulation of alpha-receptors, causing vasoconstriction and hypertensive crisis.

Atenolol is the incorrect answer. Atenolol is a beta-blocker and will cause unopposed stimulation of alpha receptors.

Bisoprolol is the incorrect answer. Bisoprolol is another beta-blocker - like atenolol, it can risk causing a hypertensive crisis.

Nicorandil is the incorrect answer. Nicorandil is a treatment option for lowering blood pressure but is not specifically used to treat hypertension secondary to a phaeochromocytoma.

Spironolactone is the incorrect answer. Spironolactone is used to treat hypertension but as above, it is not a targeted treatment for hypertension due to a phaeochromocytoma.

👍 👎 Discuss (1) Improve

Next question

Phaeochromocytoma ★

Phaeochromocytoma is a rare catecholamine secreting tumour. About 10% are familial and may be associated with MEN type II, neurofibromatosis and von Hippel-Lindau syndrome

Basics
- bilateral in 10%
- malignant in 10%
- extra-adrenal in 10% (most common site = organ of Zuckerkandl, adjacent to the bifurcation of the aorta)

Features are typically episodic
- hypertension (around 90% of cases, may be sustained)
- headaches
- palpitations
- sweating
- anxiety

Tests
- 24 hr urinary collection of metanephrines (sensitivity 97%*)
- this has replaced a 24 hr urinary collection of catecholamines (sensitivity 86%)

Surgery is the definitive management. The patient must first however be stabilized with medical management:

- alpha-blocker (e.g. phenoxybenzamine), given before a
- beta-blocker (e.g. propranolol)

*BMJ 2012; 344 doi: http://dx.doi.org/10.1136/bmj.e1042 (Published 20 February 2012)

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PHEOCHROMOCYTOMA

Phaeochromocytoma

Townsend Teaching - YouTube 👍 3 👎 0

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Score: **52.5%**

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A 40-year-old woman attends the endocrinology clinic with a 4-week history of intermittent headaches and milky discharge from her nipples. Over the last 5 months, she also reports absent menstrual periods despite usually having a regular 28-day cycle. There is no past medical history of note and she has no allergies.

Laboratory tests:

Thyroid-stimulating hormone (TSH)	0.5 mU/L	(0.5-5.5)
Free thyroxine (T4)	7.5 pmol/L	(9.0 - 18)
Adrenocorticotrophic hormone	6 ng/L	(0 - 47)
IGF-1	10 nmol/L	(12.4 - 30.3)
Prolactin	6000 IU/mL	(59 - 619)
β-HCG	< 1 u/L	(< 1)

MRI head: there is a visible pituitary adenoma of approximately 1.5cm in size causing compression of the optic chiasm.

Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

- ○ Bromocriptine ×
- ○ Dexamethasone ×
- ○ Domperidone ×
- ○ Stereotactic radiotherapy ×
- ○ Trans-sphenoidal surgery ×

Submit answer

Reference ranges

Score: **52.5%**

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Question 100 of 101 ✗

A 40-year-old woman attends the endocrinology clinic with a 4-week history of intermittent headaches and milky discharge from her nipples. Over the last 5 months, she also reports absent menstrual periods despite usually having a regular 28-day cycle. There is no past medical history of note and she has no allergies.

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MRI head: there is a visible pituitary adenoma of approximately 1.5cm in size causing compression of the optic chiasm.

Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

🟩	Bromocriptine	52%
	Dexamethasone	1%
	Domperidone	1%
	Stereotactic radiotherapy	1%
🟥	Trans-sphenoidal surgery	46%

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

Important for me   Less important

Given the history of amenorrhoea and galactorrhoea and the blood results demonstrating an elevated prolactin level with low-normal thyroid, IGF-1 and ACTH levels, this patient has a diagnosis of prolactinoma. This is confirmed by the presence of a pituitary mass of 1.5cm in size that classifies this growth as a macroadenoma. The first-line treatment for all prolactinomas (regardless of size) is the use of dopamine agonists such as bromocriptine. Dopamine suppresses the production of prolactin from the anterior pituitary gland and thus helps improve this patient's symptoms.

Dexamethasone is commonly indicated in patients with evidence of increased intracranial pressure or space-occupying lesions. Although this patient has a significant pituitary adenoma, evidence of compression of the optic chiasm and neurological symptoms including headaches, dexamethasone is not first-line in the treatment of prolactinomas. Rather, dopamine agonists are preferred.

Domperidone is a dopamine antagonist and would be contraindicated in this patient given that dopamine suppresses prolactin production and further inhibition could drive the growth of the prolactinoma.

Stereotactic radiotherapy is less popular in the treatment of such small lesions, given the risk of damage to adjacent structures and would therefore not be a first-line treatment for this patient.

Trans-sphenoidal surgery is an alternative treatment option for prolactinomas. However, this is reserved if medical treatment with dopamine agonists fails. As dopamine agonists have not yet been initiated for this patient, bromocriptine would be the correct answer.

👍 👎 Discuss (7) Improve

Next question

Prolactinoma ★

Prolactinomas are a type of pituitary adenoma, a benign tumour of the pituitary gland.

Pituitary adenomas can be classified according to:
- **size** (a microadenoma is <1cm and a macroadenoma is >1cm)
- **hormonal status** (a secretory/functioning adenoma produces and excess of a particular hormone and a non-secretory/functioning adenoma does not produce a hormone to excess)

Prolactinomas are the most common type and they produce an excess of prolactin.

Features
- excess prolactin in women
    - amenorrhoea
    - infertility
    - galactorrhoea
    - osteoporosis
- excess prolactin in men
    - impotence
    - loss of libido
    - galactorrhoea
- other symptoms may be seen with macroadenomas
    - headache.
    - visual disturbances (classically, a bitemporal hemianopia (lateral visual fields) or upper temporal quadrantanopia)
    - symptoms and signs of hypopituitarism

Diagnosis
- MRI

Management
- in the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. cabergoline, bromocriptine) which inhibit the release of prolactin from the pituitary gland

- surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PROLACTINOMA

Prolactinoma

Osmosis - YouTube 👍 5 👎 2

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Question 101 of 101

A 28-year-old man presents to the clinic for review of his familial hypercholesterolaemia. He is currently on 80mg once daily atorvastatin.

His blood results are as follows:

Hb	135 g/l	Na^+	138 mmol/l
Platelets	$322 * 10^9/l$	K^+	4.6 mmol/l
WBC	$10.5 * 10^9/l$	Urea	6.6 mmol/l
Neuts	$6.2 * 10^9/l$	Creatinine	72 µmol/l

Total cholesterol	7.5 mmol/L (normal range < 5)
LDL cholesterol	5.5 mmol/L (normal range < 3)

What treatment would you begin?

- ○ Fenofibrate ×
- ○ Nicotinic acid ×
- ○ Ezetimibe ×
- ○ Evolocumab ×
- ○ Cholestyramine ×

Submit answer

Dr. Jamil Alrajhi

Reference ranges

Score: **52%**

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Total cholesterol	7.5 mmol/L (normal range < 5)
LDL cholesterol	5.5 mmol/L (normal range < 3)

What treatment would you begin?

Fenofibrate	14%
Nicotinic acid	3%
Ezetimibe	49%
Evolocumab	33%
Cholestyramine	2%

First line management for familial hypercholesterolaemia is high dose statins. Ezetimibe is used as a second line agent

Important for me Less important

High dose statin therapy is first line for familial hypercholesterolaemia.

If serum total or low-density lipoprotein (LDL) cholesterol concentration is not appropriately controlled on statin therapy, then the second line agent ezetimibe can be co-administered with the statin. Ezetimibe acts by decreasing cholesterol absorption in the small intestine.

👍 👎 Discuss (7) Improve

Familial hypercholesterolaemia

Familial hypercholesterolaemia (FH) is an autosomal dominant condition that is thought to affect around 1 in 500 people. It results in high levels of LDL-cholesterol which, if untreated, may cause early cardiovascular disease (CVD). FH is caused by mutations in the gene which encodes the LDL-receptor protein.

Case finding:

- NICE suggest that we should suspect FH as a possible diagnosis in adults with:
    - a total cholesterol level greater than 7.5 mmol/l and/or
    - a personal or family history of premature coronary heart disease (an event before 60 years in an index individual or first-degree relative)
- children of affected parents:
    - if one parent is affected by familial hypercholesterolaemia, arrange testing in children by age 10
    - if both parents are affected by familial hypercholesterolaemia, arrange testing in children by age 5

Clinical diagnosis is now based on the **Simon Broome criteria**:

- in adults total cholesterol (TC) > 7.5 mmol/l and LDL-C > 4.9 mmol/l or children TC > 6.7 mmol/l and LDL-C > 4.0 mmol/l, plus:
- for definite FH: tendon xanthoma in patients or 1st or 2nd degree relatives or DNA-based evidence of FH
- for possible FH: family history of myocardial infarction below age 50 years in 2nd degree relative, below age 60 in 1st degree relative, or a family history of raised cholesterol levels

Management

- the use of CVD risk estimation using standard tables is not appropriate in FH as they do not accurately reflect the risk of CVD
- referral to a specialist lipid clinic is usually required
- high-dose statins are usually used first-line
- first-degree relatives have a 50% chance of having the disorder and should therefore be offered screening. This includes children who should be screened by the age of 10 years if there is one affected parent
- statins should be discontinued in women 3 months before conception due to the risk of congenital defects

AI + Q 123

Textbooks

Dr. Jamil Alrajhi

High-yield textbook

Extended textbook

Links

The FH Foundation 👍 5 👎 4

High cholesterol during preganancy

The FH Foundation 👍 7 👎 5

Simon Broom Criteria

Public Library of Science (PLoS) 👍 5 👎 3

[Cascade Screening for Familial Hypercholesterolemia (FH)]

NICE 👍 4 👎 2

[2008 Familial hypercholesterolaemia guidelines]

Suggest link

Report broken link

Media

FAMILIAL
HYPERCHOLESTEROLEMIA
Osmosis.org

Familial hypercholesterolaemia

Osmosis - YouTube 👍 10 👎 1

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